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THE RÔLE OF THE DORSAL ROOTS IN MUSCLE TONUS *

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Tonus is the steady and indefatigable contraction of the muscles required to hold the different parts of the skeleton in their proper relations in the various and constantly changing attitudes and postures of the body. It has been defined by Sherrington [1] as postural contraction. A muscle, when functioning for the maintenance of posture, tends to take and hold a given length; and when that length is altered, either by active contraction or by the passive movement of the limb, the muscle again takes and holds the new length. This is particularly evident in decerebrate rigidity. The "lengthening and shortening reactions," so well seen in decerebrate cats, have been described in numerous papers by Sherrington [2] and Brown.[3] These reactions make the limbs of decerebrate animals moldable, so that they will take and hold any degree of flexion or extension passively imposed on them. To use Sherrington's term, the limbs are plastic. This plasticity, whatever its nature and cause, is characteristic of tonically contracting muscle and is in some way related to the steadiness and indefatigability of postural contraction. These important observations were originally made by Sherrington and elaborated by him in a series of brilliant publications.

Sherrington [2] and Brown [3] have shown that this plasticity is dependent on the integrity of the dorsal roots; and on the basis of this and other related observations, the theory that tonus is a proprioceptive reflex has been elaborated. The sensory impulses from a given muscle are alone responsible for the plasticity of that muscle. This theory has recently been defended in a masterly way by Fulton; [4] but to a careful reader familiar with the related literature this book serves chiefly to show how great are the difficulties in fitting some of the observed facts to the theory.

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1. Sherrington, C. S.: Brain **38**:191 and 205, 1915.
2. Sherrington, C. S.: Quart. J. Exper. Physiol. **2**:109, 1909; footnote 1.
3. Brown, Graham: Proc. Roy. Soc. **77**:145, 1913.
4. Fulton, J. F.: Muscular Contraction and the Reflex Control of Movement, Baltimore, Williams & Wilkins Company, 1926.

That the dorsal roots play an important rôle in muscle tonus cannot be debated. But the proprioceptive reflex theory, which has been elaborated to interpret this rôle, does not adequately account for the conservation of energy and freedom from fatigue characteristic of tonic contraction; and additional far-reaching assumptions must be made in order to explain, on this basis, the weakness or absence of action currents in such contractions. These features of postural contraction will be discussed in a subsequent section of this paper. In explaining the lengthening and shortening reactions as reflexes, the proprioceptive reflex theory is stretched to the breaking point.

In Sherrington's [5] own words:

> The "shortening reaction," if reflex, is a tonic reflex, for its result on the muscle is an increased tonic shortening. There are two ways in which such a reflex might be brought about. (1) The passive relaxation of the muscle when the knee is extended by external manipulation might cut short a stimulus which was keeping the muscle elongated by reflex inhibition. But as pointed out above, there seems no firm ground for assuming that persistent inhibitory stimulus is then at work. (2) The passive relaxation of the muscle might itself act as a stimulus to intramuscular receptors, productive of reflex tonic excitation of the muscle. The latter view postulates the possession by the muscle of afferent nerve fibres which can excite it to reflex contraction. Now the conditions essential for decerebrate rigidity seem to prove that such afferent fibres are possessed by the muscle. A difficulty regarding them is that they are not revealed by direct stimulation, either mechanical or electrical, of the vastocrureus nerve itself, although it is in that nerve that they must lie.

More recently, Liddell and Sherrington [6] have made observations which they regard as direct evidence of sensory fibers in the muscles, stimulation of which causes a reflex tonic contraction. But these fibers are stimulated by stretching the muscle and could not be responsible for the shortening reaction which occurs when the muscle is passively relaxed.

One may go further and ask how the lengthening reflex can be accounted for, if tension is the adequate stimulus for these stretch afferents. Passive flexion, by stimulating the stretch afferents of the extensor muscles, should cause an increase in their tonic contraction, not a reflex relaxation as would be required if one is to explain the lengthening reaction in this way. Fulton [4] has tried to overcome this difficulty by assuming that reflex relaxation occurs when the force exerted in flexing the joint is sufficiently violent to stimulate the pain fibers in the stretched extensor muscles. This assumption has no basis in fact, however, for, as Sherrington [5] has shown, the lengthening

5. Sherrington (footnote 2, first reference).

6. Liddell, E. G. T., and Sherrington, C. S.: Proc. Roy. Soc. B. **96**:212, 1924.

reaction can best be demonstrated when decerebrate rigidity is not of high grade and when a traction of 40 or 50 Gm. is sufficient to flex the joint.

While I do not see how the lengthening and shortening reactions can be explained by the proprioceptive reflex theory of tonus, I can agree fully with Sherrington [5] when he says:

> From the foregoing it is clear that both "lengthening reaction" and "shortening reaction" of vastocrureus are indissolubly connected with afferent nerve fibres distributed in that muscle itself. The functioning of the intrinsic proprioceptive arc, i. e., the proprioceptive arc of vastocrureus itself, is essential to these reactions, and no other arc is essential. So much seems proved. Yet, to connect these reactions with the functioning of the intrinsic proprioceptive arc, and to show, as above, that that arc is indispensable to them, and uniquely indispensable, does not prove that the reactions themselves are strictly reflex. It is conceivable that the functioning of that arc confers upon the muscle fibres a state, e. g., tonus, with properties which render the muscle fibres plastic in the above-mentioned sense, so that when subjected to mechanical changes such as shortening and stretching, they retain the shortening and lengthening impressed upon them.

In view of all this, the antidromic conduction of tonic impulses over the dorsal root, as postulated by Frank,[7] offers an attractive field for speculation. Such tonic impulses might cause a gelling or increase in viscosity of the muscle fibers fixing them at any given length—and this would readily account for the lengthening and shortening reactions. Yet to keep on solid ground one must admit that there is no convincing evidence for such antidromic conduction of tonic impulses and no histologic evidence of the necessary innervation of large numbers of skeletal muscle fibers by nerve fibers of dorsal root origin.

In spite of all of the work which has been done to determine its nature and cause, it must be realized that muscle tonus presents an unsolved problem. A theory cannot be considered satisfactory which does not provide a mechanism for maintaining posture without fatigue and with a minimum expenditure of energy, by a type of contraction which is more or less free from action currents. This problem is of great importance not only because normal muscle tonus is essential for smoothness of motion and steadiness of posture, but because the stiffness caused by the abnormally increased tonus, which results from certain lesions of the nervous system, is a frequent cause of disability.

CONCEPTIONS OF THE HOLDING MECHANISM RESPONSIBLE FOR PLASTICITY IN TONICALLY CONTRACTING MUSCLE

The necessity for postulating some type of holding mechanism was recognized by Langley [8] when he said that certain forms of prolonged

7. Frank, E.: Berl. klin. Wchnschr. **57**:725, 1920; Arch. f. exper. Path. u. Pharmakol. **90**:149, 1921.

8. Langley, J. N.: J. Physiol. **57**:LXX, 1923.

contraction, which cause little or no fatigue and little or no chemical change, may be due to a continuous activity of the nerve cells causing a movement of ions and, thus, an increase of muscular tension, which is then maintained without further change. Bayliss [9] said that the economy of effort in decerebrate rigidity is extraordinary. Roaf [10] found that metabolism did not increase in decerebrate rigidity and that the formation of carbon dioxide was not decreased by section of all of the nerves going to the limbs in decerebrate cats. Grafe [11] could not detect an increase in metabolism during states of hypertonus. Cases of tetanus, postencephalitic rigidity and a series of other organic nervous disorders were investigated.

The indefatigability of postural contractions has recently been emphasized by Magnus.[12] After unilateral extirpation of the labyrinth in the rabbit, turning of the neck results which evokes the normal tonic neck reflex, one forelimb being extended, the other flexed. The turning of the head persists as long as the animal lives, and the tonic neck reflexes at least for several months. "We are accustomed," Magnus says, "to believe that muscular action is liable to fatigue, and this, of course, is true for movements and especially for movements performed against resistance. But muscular action concerned in keeping some part of the body in constant and unchanging position gives rise to much less fatigue, and the attitudinal tonic reflexes evoked from the head appear to be practically indefatigable."

The special nature of tonic contractions is evidenced by the fact that they are accompanied by weak action currents, which in certain types of tonic contraction such as tetanus rigidity may be entirely absent (Liljestrand and Magnus [13]). Wertheim-Salomonson [14] asserted that in all true forms of muscle tonus and hypertonus action currents could not be detected. Since they are present, though weak, in decerebrate rigidity he regarded this condition as a tetanic state. He said: "The absence of a typical action current seems the one and only means of differentiating between a tetanic and a tonic spasm or contraction." Doubtless, he overstated his case, for weak action currents have been detected in most forms of tonic contraction. The subject was recently discussed by Fulton,[4] and an effort was made to harmonize the facts with the conception that tonus is due to an all-or-none contraction of muscle fibers activated as a proprioceptive reflex. The literature is too voluminous and technical to be considered here. The present

9. Bayliss, William: Livre jubilaire du Prof. Ch. Richet, 1912, p. 471.
10. Roaf, H. E.: Quart. J. Exper. Physiol. **5**:31, 1912.
11. Grafe, E.: Deutsches Arch. f. klin. Med. **139**:155, 1922.
12. Magnus, R.: Proc. Roy. Soc. B. **98**:339, 1925.
13. Liljestrand, G., and Magnus, R.: Arch. f. d. ges. Physiol. **176**:168, 1919.
14. Wertheim-Salomonson: Brain **38**:193, 1921.

status of the problem, however, has been well stated by Riesser,[15] who said that the complete absence of oscillatory action currents makes the tonic nature of a muscle contraction certain, but that the presence of slight action currents does not exclude the predominantly tonic character of a contraction.

All-or-None Contractions.—Contractions of muscle fibers conforming to the all-or-none law and activated through the proprioceptive system by afferent impulses coming from the muscles themselves are believed by Fulton [4] to form an adequate basis for an explanation of tonic contractions. Forbes [16] is the author of an ingenious hypothesis:

> Possibly a group of fibers in a shortened state, either by virtue of their own contraction or of those about them, sends to a limited number of motor neurons the requisite proprioceptive impulses to establish reflex contraction. The muscle fibers thus excited may be different from the first group. Synaptic fatigue releases these muscle fibers before they become subject to fatigue; but meanwhile their contraction has reflexly evoked that of a third group. And so the fibers may take up the load in rotation and, for some reason, by this means attain an economy otherwise impossible.

This theory would, perhaps, account for the absence of fatigue; and, because the action currents of the individual fibers would be out of phase, it furnishes the best explanation yet offered for the weakness or absence of action currents; but it is not at all clear in what way it would bring about an economy of effort. In any event one must not forget that it is only a hypothesis. By no means all electrophysiologists are prepared to admit that this theory forms an adequate explanation of the weakness of action currents in most tonic contractions and their complete absence in tetanus rigidity.

Certainly it cannot be said that the postural function of skeletal muscle has as yet been satisfactorily accounted for on the basis of the all-or-none law. Other possibilities must therefore be kept in mind.

Special Muscle Fibers.—The contraction of the fine muscle fibers which are rich in sarcoplasm and found in great numbers in red muscle was considered by Hunter [17] as the basis of tonus. According to him, the coarse fibers, which are rich in myofibrils and which make up the bulk of white muscles, are innervated by myelinated motor nerve fibers; while the fine fibers, which are rich in sarcoplasm and are found in large numbers in red muscle, are innervated by unmyelinated sympathetic fibers. It is thought that the coarse fibers function in voluntary and reflex activity and are responsible for contractile tonus.

15. Riesser, O.: Klin. Wchnschr. **4**:53, 1925.
16. Forbes, A.: Physiol. Rev. **2**:361, 1922.
17. Hunter, J. I.: Brit. M. J. **1**:197, 251, 298, 350 and 398, 1925.

The fine fibers, the contraction of which resembles that of smooth muscle, are considered capable of holding any length passively imposed on them and are responsible for plastic tonus. Hunter and his co-workers have not been able to present convincing evidence in favor of this theory. A review of the literature on red and white muscles and their relation to tonus is not required here, since this ground has recently been covered in detail by Needham.[18]

Sarcoplasmic Contraction.—A slow tonic contraction of the sarcoplasm was postulated by Bottazzi [19] in 1897, and was thought to support the myofibrils in their quick contractions. Most of the advocates of the sympathetic origin of tonus have had recourse to this sluggish contraction of the sarcoplasm as the holding mechanism responsible for tonus. The literature on the sympathetic innervation of skeletal muscle is too voluminous to be reviewed at this point. Cobb,[20] Coman,[21] and Fulton [4] have recently surveyed this field in some detail and have not found any convincing evidence in favor of this theory. In collaboration with Hinsey, I [22] have demonstrated that the sympathetic nervous system does not take an appreciable part in the production of decerebrate rigidity or the rigidity caused by tetanus toxin.

Antidromic Conduction Along Dorsal Root Fibers.—However, the sympathetic nervous system is not the only possible source of special tonic impulses. Frank [7] believed that antidromic conduction along the dorsal root fibers causes the slow tonic contraction which he regarded as the basic factor in muscle tonus. A number of facts are in accord with this theory. Stimulation of sensory fibers, going to a muscle the motor nerve fibers of which have degenerated, causes a slow tonic contraction as seen in the Vulpian-Heidenhain [23] and Sherrington [24] phenomena. A similar contraction is caused in frog muscle and in denervated mammalian muscle by small doses of acetylcholine. Since this drug is a powerful parasympathetic stimulant, and since its action on muscle is depressed by scopolamine, Frank thought that these special tonic fibers of the dorsal root belong to the parasympathetic system. Gasser and Dale,[25] however, have shown that not all parasympathetic stimulants have this property of causing tonic contraction and that these drug contractions are accompanied by the production of lactic

18. Needham, D. M.: Physiol. Rev. **6**:1, 1926.
19. Bottazzi, P.: J. Physiol. **21**:1, 1897.
20. Cobb, S.: Physiol. Rev. **5**:518, 1925.
21. Coman, F. D.: Bull. Johns Hopkins Hosp. **38**:163, 1926.
22. Ranson, S. W., and Hinsey, J. C.: J. Comp. Neurol. **42**:69, 1926.
23. Heidenhain, R.: Arch. f. Physiol., 1883, supp., p. 133.
24. Sherrington, C. S.: J. Physiol. **17**:211, 1894-1895.
25. Gasser, H. S., and Dale, H. H.: J. Pharmacol. & Exper. Therap. **28**: 287; **29**:53, 1926.

acid, which is of the same dimensions as that observed in a tetanus producing equivalent tension. They conclude that there is not any ground for suggesting that this type of contraction depends on an alternative contractile metabolism, or that it corresponds to a second, parasympathetic innervation. It is significant, however, that in paralysis agitans and in the postencephalitic syndromes atropine and scopolamine tend to decrease the rigidity. And as Schäffer [26] has shown, Tiegel's contracture in man is increased by pilocarpine and physostigmine (parasympathetic excitants) and is suppressed by atropine and scopolamine (parasympathetic depressants).

Elsewhere I [27] have called attention to the fact that both nicotine and acetylcholine, when injected intra-arterially, cause a vasodilation resembling that caused by stimulation of the dorsal roots, and that they both also cause a slow tonic contraction of denervated mammalian muscle, which resembles that caused by stimulation of the sensory nerve fibers in the Vulpian and Sherrington phenomena. Since the action of these drugs on the blood vessels and the muscles is increased by denervation, the idea inevitably suggests itself that the stimulation occurs at the myoneural junction between sensory nerve fibers and muscle.

Those who have supported the idea that a special innervation, either sympathetic or antidromic, is responsible for tonic contraction have, for the most part, based their theories on Bottazzi's conception of the slow contraction and tardy relaxation of sarcoplasm. The dualistic theory of muscle function, however, can be formulated in quite a different way.

Physicochemical Changes.—A setting of the muscle fibers due to a gelling or increase in viscosity caused by special tonic impulses must be thought of as a possibility. "According to this theory, a muscle fiber contracts (due to a motor impulse) and then becomes fixed in this contracted state. This fixation produces the stiffness characteristic of the limbs of decerebrate animals—a stiffness which resembles that of rigor mortis." When I made this suggestion, in 1926, I thought that it was original, but later I learned that this theory was definitely formulated by von Trzecieski [28] in 1905. He thought that impulses, which travel centrifugally over the dorsal roots, inhibit the relaxation of muscle, and thus hold it in the state of contraction. As a corollary he assumed that these impulses, by tending to fix the muscles at varying lengths, would retard and moderate the contractions initiated by impulses reaching the muscles over the ventral root fibers, and in this way contribute to the smoothness of movement.

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26. Schäffer, H.: Arch. f. d. ges. Physiol. **185**:42, 1920.
27. Ranson, S. W.: J. Comp. Neurol. **40**:1, 1926.
28. Von Trzecieski, A.: Arch. f. Physiol., 1905, p. 306.

In spite of some serious difficulties encountered in the accepting of Trzecieski's theory, the idea is deserving of the most careful consideration. It is in accord with most of the known facts concerning the relation of the dorsal roots to muscle tonus. In some respects it serves as a more adequate explanation of plasticity, the lengthening and shortening reactions, decerebrate rigidity and the resistance of tonic muscle to passive stretching (the "myotatic reflex") than does the alternative theory that tonus is a proprioceptive reflex.

Some evidence in favor of this theory was presented by me in 1926. I found that the application of weak solutions of nicotine to the spinal ganglia of the lumbosacral plexus reduced decerebrate rigidity in the corresponding hind limb. Since after the rigidity had been greatly reduced, the flexion and crossed extension reflexes could be obtained by pinching the toes of either foot, and since these reflexes were often

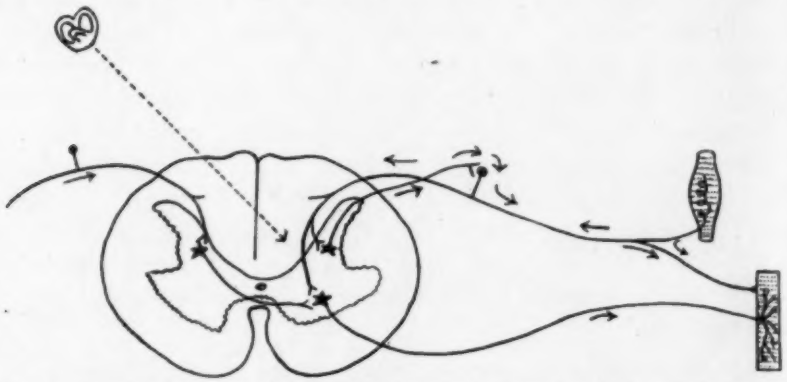

Fig. 1.—Some of the possible conduction paths concerned in muscle tonus.

more brisk on the nicotinized side, there is not any reason to suppose that the nicotine impaired the conductivity of the motor or sensory fibers. But, since it seemed to block the passage of tonic impulses through the ganglion, and since the work of Dogiel [29] and others has indicated that most spinal ganglion cells are surrounded by pericellular plexuses like those seen in sympathetic ganglia, the suggestion was made that the tonic impulses passed through synapses in the spinal ganglia in the manner indicated in figure 1. Of course, such indirect evidence as this is not convincing, and more direct proof that tonic impulses reach the muscles through the dorsal roots is required. There is, however, enough suggestive evidence to justify a thorough-going investigation of tonus along lines suggested by the hypotheses of Trzecieski and Frank.

I do not regard these two theories as mutually exclusive. Slow contraction and setting may be only different aspects of the same change

29. Dogiel, A. S.: Der Bau der Spinalganglien, Jena, G. Fischer, 1908.

occurring in the sarcoplasm. According to both theories, the change is initiated by impulses traveling antidromically over the dorsal roots.

The chief difficulty in the acceptance of either of these hypotheses is that there is not any histologic evidence that the sensory fibers give off side branches which might serve for the tonic innervation of the ordinary muscle fibers. Some such mechanism as that shown in figure 1 would be needed for the antidromic conduction of tonic impulses along the sensory fibers to the muscles. But Hinsey,[30] who has made a careful study of the innervation of skeletal muscle with this particular problem in mind, has failed to find evidence of the innervation of any considerable number of skeletal muscle fibers by nerve fibers whose cells of origin are located in the spinal ganglia. Certainly the intrafusal fibers of the muscle spindles are not numerous enough to provide by their contraction for the tonic shortening of skeletal muscle.

That the dorsal roots play an important rôle in muscle tonus cannot be debated. None of the theories which have been advanced to explain this rôle, however, is entirely satisfactory. In fact, just what effect section of the dorsal roots has on muscle tonus is not altogether clear, as will be made evident by the paragraphs which follow.

RESULTS OF CUTTING THE DORSAL ROOTS

Section of a large number of dorsal roots produces a temporary depression of spinal cord function not unlike that caused by transection of the cord. Brooks [31] found that there was a parallelism between the changes in the reflex irritability of the cord following the transection of the spinal cord itself, and those following transection of the sensory nerve roots. Dogs were allowed to recover from the shock of transection of the cord and exposure of the lumbosacral nerve roots. After twenty-four hours, when the crossed extension reflex had become brisk, the wound was reopened without an anesthetic and four dorsal roots (the last thoracic and first three lumbar) were cut on the right side. After this the crossed extension reflex was again abolished for nearly two hours. A temporary depression of the crossed extension reflexes followed section of the dorsal root in cats, but it was not so long continued as in the dog.

Forbes and Cattell [32] found it difficult to evoke a crossed extension reflex when the dorsal roots had just been cut in the decerebrate preparation; the reflex was much less impaired when the preparation had been deafferented aseptically several days before. But it seemed to them

30. Hinsey, J. C.: J. Comp. Neurol., 1927, to be published.

31. Brooks, C.: Am. J. Physiol. **27**:212, 1910-1911.

32. Forbes, A., and Cattell, M.: Am. J. Physiol. **70**:140, 1924.

more likely that the cause of the failure of the reflex immediately after severance of the dorsal roots lay in the disturbance of the spinal cord, which inevitably occurs in such an operation, rather than in the mere interruption of the afferent path.

These experiments show that after the dorsal roots supplying one limb have been cut, there is a depression not only of the tonus of that limb but of the crossed extension reflex, the arc of which has not been interrupted. Great caution must, therefore, be exercised in using the disappearance of tonus after dorsal root section as an argument that it is a proprioceptive reflex. The crossed extension reflex returns after the shock disappears; but so does the tonus of the deafferented muscles (Liljestrand and Magnus [13]).

That structural changes occur in the cells of the spinal cord following section of the dorsal root has been shown by Warrington,[33] Braeunig [34] and Lapinsky.[35] These changes include chromatolysis of spinal cord cells, especially those of Clarke's column and the posterolateral group of cells in the anterior horn. Lapinsky thought that these changes accounted for the pseudoparesis which often results from section of the dorsal root, but this seems doubtful for reasons which will be presented later. It is reasonable to assume, however, that such alterations of the cells of the spinal cord are related to the more or less transient flaccidity and loss of reflexes which are seen immediately after section of the dorsal root. Whether these changes are due to the vascular disturbances, resulting from section of the small arteries accompanying the dorsal roots, or to some other cause remains undetermined. But it is clear from the work of the three investigators mentioned and from the degenerations seen by Trendelenburg [36] and me, to be described later, that the dorsal roots cannot be cut without damage to the spinal cord. All of these experiments have been carefully performed, and the results are not due to mechanical insult to the cord or to infection. It is therefore clear that unless the results are controlled by postmortem evidence, and a histologic examination of the spinal cord shows it to be normal, one cannot be sure that the results of section of the dorsal root are due to the cutting off of afferent impulses.

Experimental section of the dorsal roots has led to inconsistent results, many of which are hard to explain. Earlier workers, after section of all or nearly all of the roots supplying a limb, obtained results suggesting a paresis of the anesthetic leg. This is well illustrated

33. Warrington, W. B.: J. Physiol. **23**:112, 1898-1899.

34. Braeunig, K.: Arch. f. Physiol., 1903, pp. 251 and 480.

35. Lapinsky, M.: Arch. f. Psychiat. **42**:869, 1907.

36. Trendelenburg, W.: Arch. f. Anat. u. Physiol., Physiol. Abt., 1906, supp., p. 231.

by the experiments of Mott and Sherrington [37] who cut, in monkeys, the whole series of sensory roots sending fibers to one forelimb or one hind limb.

> From the time of performance of the section onwards, as long as the animal may be kept, the movements of the hand and foot are practically abolished; the movement of grasping, which is so frequent and useful to the monkey, both with the hand and foot, never occurs at all in our experience. On the other hand, the movements at the elbow and knee, and especially the movements at the shoulder and hip, are much less impaired. The fore limb hangs from the shoulder partially flexed at the elbow; the hind limb is flexed at hip and knee. As the animal runs about it does not attempt to use the leg; the fore limb swings helplessly, with flexion at elbow and wrist and adduction at shoulder, in much the same position as if carried in a sling. The hind limb looks as if it were being held up so as to be kept off the ground while the animal runs on three legs; we are inclined to think that this appearance is deceptive, and that the position results from an equilibrium of the action of the muscles, in which purposive action on the part of the animal does not play a rôle. In the case of the lower limb, after two or three months, the constant position of flexion of hip and knee, on two occasions, gradually induced a change in the muscles of the thigh, which prevented hip and knee being properly extended, even by passive stretching.

Von Korniloff [38] obtained similar results with dogs. After section of seven dorsal roots in the lumbar enlargement, he observed a complete paralysis of the movement of the toes and foot; the other parts of the limb were only paretic.

After unilateral section of the dorsal roots in the lumbosacral region, Bickel [39] found that his dogs never used the leg regularly in walking. The anesthetic leg would be used for a step or two and then dragged for a few steps as if it had been forgotten. This question of the apparent paresis of a completely deafferented leg will be discussed in a later section of this paper.

When the section of the dorsal roots does not result in a pseudoparesis, the movements of the deafferented limb are awkward, excessive and poorly controlled because sensory impulses coming from the active muscles no longer take part reflexly in their control. As might have been expected, ataxia has been seen by most investigators. I have seen it in my experiments, but will make no further mention of it because it is too obvious to need emphasis.

One would also expect atonia to result from section of the dorsal root, but on this point the evidence is not so clear. Hering [40] always

37. Mott, F. W., and Sherrington, C. S.: Proc. Roy. Soc. **57**:481, 1895.

38. Von Korniloff, A.: Neurol. Centralbl. **15**:924, 1897; Deutsche Ztschr. f. Nervenh. **12**:199, 1898.

39. Bickel, A.: Arch. f. d. ges. Physiol. **67**:299, 1897.

40. Hering, H. E.: Neurol. Centralbl. **15**:1077, 1897.

found decreased resistance to passive movement in the deafferented limbs of frogs, dogs and apes. Liljestrand and Magnus [13] found that, during the first few days after section of the dorsal roots of one brachial plexus, the triceps was entirely flaccid but retained the capacity for active movement. After about one week, the muscle again acquired tonus which increased in time. In this and similar experiments the question is raised whether the first toneless condition of the muscles is caused exclusively by the section of the sensory fibers or by the shock of the operation, and whether the returning tonus is due to a disappearance of this shock. Liljestrand and Magnus believed that their results showed clearly that the proprioceptive impulses from the muscles play the chief rôle in the normal animal in the production of muscle tonus. After their interruption, the muscle becomes entirely or almost entirely flaccid; and when tone returns some time after section of the dorsal roots, this is due to an increase in the irritability of the corresponding motor centers for afferent impulses from other sources.

Foerster [41] advocated cutting the dorsal roots as a cure for spastic paralysis. The operation has its justification in the generally accepted idea that the dorsal roots are responsible for muscle tonus and that when they are divided the deafferented muscles become atonic. Steinke [42] has summarized the results of 132 reported operations by Foerster's method for various forms of spastic paralyses. Four of the patients were recorded as cured and 97 as improved. There were 20 deaths, and 11 patients did not show improvement. The results of the operation have, on the whole, been rather disappointing, and it is not widely employed.

The first experiment concerning the relation of the dorsal root to muscle tonus was conducted by Brondgeest [43] in 1860. He cut the dorsal roots for one hind leg and then the spinal cord near the medulla. He then supported the frog by a thread through the nose and found that the control leg was drawn up in a more flexed posture than the deafferented leg. From this experiment he drew the conclusion that voluntary muscle exhibits tonus in the form of an enduring contraction, owing to a continuous discharge from the nerve centers, and that the existence of this tonus is intimately connected with the integrity of the sensory nerves. Twenty-five years later, Mommsen [44] repeated Brondgeest's experiment with identical results. It is important to note, however, that neither investigator allowed time for the frogs to recover from the operation before conducting the tests; Brondgeest apparently made the tests without delay, and Mommsen waited for only a few

41. Foerster: Deutsche Ztschr. f. Nervenh. **58**:151, 1918.
42. Steinke, C. R.: Surg. Gynec. Obst. **27**:55, 1918.
43. Brondgeest, P. J.: Arch. f. Anat. u. Physiol., 1860, p. 703
44. Mommsen, J.: Virchows Arch. f. path. Anat. **101**:22, 1885.

hours at most, after the section of the roots. It should be mentioned also in this connection that Brooks [31] found that, immediately after dorsal root section, the dog, cat and frog showed a marked depression of spinal cord function, as tested by the crossed extension reflex; but the rabbit, guinea-pig, pigeon, chicken and turtle show little or none. It is therefore evident that the frog, in spite of its low position in the zoologic scale, is not well suited for such an experiment.

Brondgeest's work is well known, but the much more significant work of Bickel, published in 1897, has apparently escaped the attention of recent workers interested in muscle tonus. Bickel allowed his frogs to recover from the operation, and found that the symptoms remained the same for weeks and months. If, after the recovery from the section of all of the sensory roots for both hind legs, the frog is suspended in the water, both hind legs are drawn up to the same extent; but, when the dorsal roots have been cut on only one side, the leg on that side is drawn up more than the normal one. These observations were repeated on many frogs. It was first necessary, of course, to keep the frog suspended until it came to rest in the water. These results cannot easily be harmonized with the conception that tonus is a proprioceptive reflex or with the idea that section of the dorsal roots necessarily produces atonia.

Trendelenburg [45] cut the dorsal roots supplying one or both wings in pigeons and allowed the birds to recover from the operation. After a unilateral section, the posture of the wings was the same on both sides when the animal stood or walked, and also when it hung with its head downward. Even after the bilateral operation, the posture of the wings was normal while the bird stood or walked or hung with its head downward. He concluded that the tonus responsible for normal posture of the wing is not called forth reflexly from the wings. Hunter,[46] and Kuntz and Kerper,[47] working with the domestic fowl, found that after section of the dorsal roots of the lower four cervical nerves, which send their fibers into the brachial plexus, plastic tonus remained and, if one can judge from the accounts of their observations, this tonus was even exaggerated, because the wing tended to remain in any position in which it was passively placed. This plastic tonus could be abolished by section of the dorsal root of the first thoracic nerve, which, according to them, contains the sympathetic afferent fibers from the muscles. These observations were made at a time when the authors were involved in an unfortunate controversy; but whatever one may think of this recent work, the original experiments of Trendelenburg

45. Trendelenburg, W.: Arch. f. Physiol., 1906, p. 1.
46. Hunter, J. I.: Surg. Gynec. Obst. **39**:721, 1924.
47. Kuntz, S., and Kerper, A. H.: Am. J. Physiol. **76**:121, 1926.

must be given great weight. It should be noted in this connection that Trendelenburg found that the brachial plexus in pigeons received sensory fibers from six nerves instead of four, as described by Hunter for the domestic fowl. In some of Trendelenburg's experiments he cut eight roots, including one above and one below the brachial plexus. He did not notice any special significance of the first thoracic dorsal root for tonus; at any rate, he did not mention it in his paper.

Trendelenburg noticed an exaggerated reflex activity of the deafferented wing, an observation which is of the greatest importance in the interpretation of the phenomena seen in the completely deafferented mammalian limb. He found that the wing reflexes, which occur when the bird is suddenly moved by lowering the rod on which it sits, were more active on the deafferented side, were also more extensive and could be induced by stimuli which did not affect the normal wing. This was interpreted as indicating that by section of the dorsal roots a normal reflex inhibition had been abolished. According to Trendelenburg, this reflex inhibition must arise from the active wing itself. One difficulty with this idea is that when the rod on which the bird rests is moved only slightly downward the wing reflex appears only in the deafferented wing. Here, then, the reflex in the normal wing must be inhibited from the beginning, which would indicate a great sensitivity of this reflex dampening apparatus. For this reason, Trendelenburg assumed that a continuous depression of the central paths is brought about through the dorsal roots, which disappears after section of the dorsal root. On this theory, one might expect to find an overaction of the dominant muscles of a deafferented limb as a result of the cutting off of the inhibitory afferent impulses, which in the intact animal reach the spinal cord from the active muscles. This does, in fact, occur.

In my experiments on cats I have observed phenomena which might easily be interpreted as representing a paresis of the deafferented limb (fig. 4 *B*), but which are, in fact, due to an overaction of the extensor muscles which prevent normal flexion of the limb. Whether the pseudoparesis described by earlier workers was of this same nature can only be conjectured; but abundant evidence is found in the literature that complete deafferentation of a limb often causes an overaction of the dominant muscles of the affected extremity, amounting in some instances to contracture.

This is well illustrated by the quotation given from the paper by Mott and Sherrington. Their monkeys carried the deafferented arm in much the same position as if it were in a sling, i. e., flexed at the elbow and wrist and adducted at the shoulder. The deafferented hind limb of other monkeys was held up off the ground in a position of flexion; and in two instances this constant position of flexion of the

hip and knee finally resulted in contracture, which prevented the hip and knee being properly extended even by passive stretching.

A similar condition was seen by Bickel [39] in the frog. He found that if all sensory roots for both hind legs were cut and time was allowed for recovery from the operation, the plantar surfaces of the feet were commonly bent upward so that the dorsal side of the feet often lay directly on the frog's back. After recovery from section of the sensory roots for one forelimb, the deafferented extremity was extended forward and lateralward, as if in a cramp. Sometimes the cramp persisted as long as the animal was quiet but vanished when the limb was used for swimming or jumping; in other frogs the cramp occurred only when a movement was attempted but was not in evidence when the animals were quiet; in other instances the cramp was continuous and the foreleg could not be bent.

In two dogs, in which the sensory roots for one hind leg were cut, prolonged tonic shortening of the extensor muscles of that leg was observed. In one of these dogs the cramp persisted for four months. When the dog lay or stood in its cage, the anesthetic leg was always held in marked extension, and considerable pressure against the pad of the foot was required to flex the leg. The stiff leg was dragged behind in walking. This could easily have been mistaken for paresis. In many of his dogs with unilateral section of the dorsal roots hyperesthesia was present in the opposite leg. This, it seems to me, gives the clue to the explanation of these peculiar results. The extensor cramp was in the nature of a prolonged crossed extension reflex. This interpretation is supported by the fact that Bickel found that those dogs in which both hind legs had been deafferented showed less disturbance of function than those in which the roots were cut on only one side. There was less irregularity in walking; such disturbances as occurred were more quickly compensated after bilateral section of the dorsal roots. Bickel's explanation that since the dog could easily run on three legs it did not need to use the deafferented limb, but was forced to learn how to use the anesthetic members when the operation was bilateral and thus came more quickly to compensate for the sensory defect, does not seem to me satisfactory. It seems probable that the animals recovered more quickly after bilateral section of the dorsal root, because exaggerated crossed extension responses were thus eliminated.

Kopczynski,[48] working with monkeys, found that section of one dorsal root of the brachial plexus did not cause any change in motility. Section of many roots, but not the entire series supplying one limb, caused a temporary alteration of motility, some incoordination and

48. Kopczynski, S.: Polnisch. Arch. f. biol. u. med. Wissensch. **3**:99, 1907.

awkwardness in the use of the arm and especially of the fingers. Section of the entire series of dorsal roots supplying one arm caused a characteristic train of symptoms. The monkey held the arm as in a sling, flexed at the elbow and adducted at the shoulder, and could not use the limb to grasp its food. Voluntary movements were rare; those of the hand were even less frequent than those of the arm. These movements did not begin until the third week after the operation. These results correspond closely with those obtained by Mott and Sherrington [37] and clearly indicate a continuous contraction of the flexor muscles resulting in the maintenance of a sustained and characteristic posture. In these monkeys section of the dorsal root appears to have resulted in an exaggerated tonus of the flexor muscles, just as in Bickel's dogs there was evidence of an exaggerated tonus of the deafferented extensor muscles.

TECHNIC

In adult cats the dorsal roots of the right lumbosacral plexus were cut intradurally. Usually the entire series from the third lumbar to the third sacral, inclusive, were divided. In one instance the roots were cut on the left instead of the right side. In this way one hind leg was completely deafferented. The femoral and obturator nerves are formed in the cat from the fourth, fifth and six lumbar nerves and the sciatic from the sixth and seventh lumbar and the first and sometimes, also, the second sacral nerves. The quadriceps femoris, which is the muscle chiefly responsible for maintaining an extended posture of the limb, receives its afferent fibers from the fourth, fifth and sixth lumbar nerves (Owen and Sherrington).[49]

Under ether anesthesia and with aseptic technic the spines and laminae of the lower lumbar vertebrae and the dorsum of the sacrum were exposed. The muscles attached to the articular processes were cut as close as possible to the bone, care being taken not to cut the small arteries which lie close to the anterolateral border of these processes. Then the spines and the dorsal or inferior member of each pair of articular processes were removed. The laminae were then cut away with small rongeur forceps. The dura can be exposed with little loss of blood. Venous bleeding from the bone stops at once on the application of a small piece of muscle to the bleeding surface.

The dura was opened lengthwise with sharp pointed scissors. A transverse cut was made on each side from the upper end of the longitudinal incision in order to prevent the margin of the incised dura from producing pressure on the cord. The dorsal roots were raised with a probe and cut close to the spinal cord with scissors, beginning with the lower sacral roots. It was easier to divide the third and fourth lumbar roots with a curved knife.

In order to identify the roots, one starts with the seventh lumbar, which makes its exit from the vertebral canal at the level of the crest of the ilium. It is usually the largest of the series, though sometimes the first sacral is a little larger. Starting with this easily recognized root, the others can be readily identified by number.

The arterioles, which accompany the dorsal roots, are too small to be seen except for one or two large ones, which usually run with the fourth or fifth

49. Owen, A. G. W., and Sherrington, C. S.: J. Physiol. **43**:232, 1911.

lumbar roots and may be on either the right or the left side. When it becomes necessary to cut one of these, some hemorrhage occurs—otherwise, the section of the roots gives rise to little bleeding.

The roots were laid back over the cord. Attempt was not made to close the dura nor to suture the muscles, because of the danger of producing pressure on the cord. The muscles were brought together, and dead space was obliterated by the line of sutures in the lumbodorsal fascia. The incision in the skin was closed with suture clips. The identification of the roots which had been cut was checked at autopsy, until I became confident of my ability to identify them correctly at the operation.

While section of the dorsal roots is a relatively easy operation, removal of the spinal ganglia is extremely difficult, because of hemorrhage. This can be greatly reduced, however, if one bears in mind the position of the blood vessels. In removing the additional bone necessary to expose the ganglia, one must leave in place the anterior (cephalic) border of the ventral (superior) articular process near which the dorsal branch of the segmental artery is usually situated. The large vein, which lies ventral to the nerve and ganglion, is likely to be torn if undue traction is exerted on the dorsal root. By exerting slight traction on the divided dorsal root and using a sharp pointed scalpel to dissect between the ventral root and the ganglion, the latter can be removed without damage to the motor fibers.

In every instance but two, the wounds healed without infection. These two infected cats were, of course, excluded from the series.

EXTENSOR RIGIDITY OF DEAFFERENTED LIMBS

When a cat recovers from the anesthetic, following section of the entire series of dorsal roots supplying one hind leg, the anesthetic leg is flaccid, and, although it is not entirely paralyzed, its movements are more limited than those of the normal leg. The deafferented muscles are atonic. Within forty-eight hours, however, a pronounced change in the condition of the limb usually occurs. An overaction of the deafferented extensor muscles develops, which may or may not be accompanied by an increased resistance to passive flexion. This sequence of events is illustrated by the following extracts from the protocol of cat 7

Cat 7.—December 11, 3:10 p. m.: The dorsal roots were cut on the right side from the fourth lumbar to the third sacral, inclusive.

5 p. m.: The cat could walk, using the left hind leg perfectly. The right was weak and ataxic and would not support the weight of the body. It could be passively moved about, flexed and extended without the normal resistance.

Dec. 12, 9 a. m.: There was considerable stiffness of the right hind leg, which became maximally extended at the knee and ankle whenever the cat attempted to move. The right thigh was adducted, so that when an attempt was made to set the cat on its feet the right foot was placed in front and to the left of the left foot, and the cat fell on the right hip. When supported by a hand under the chest and abdomen the cat held the left hind leg semiflexed, but the right was held in full extension. This position of extension was maintained with moderate force. There was definite though moderate resistance to passive flexion.

Although, as shown in the preceding protocol, cat 7 was able to walk on three legs within two hours after the operation, it was unable to stand the next day. It is usually ten days or two weeks before the cats are able to stand, because, although the control of the left leg is normal, the right leg is not only hyperextended but is adducted beyond the midline of the body. And for this reason, if the cat is set on its feet it falls onto the right hip and cannot flex the right leg under it to rise again. This overaction of the extensor and adductor muscles of the deafferented right leg is well illustrated in figure 2 *A*, *B*, *C* and *D*,

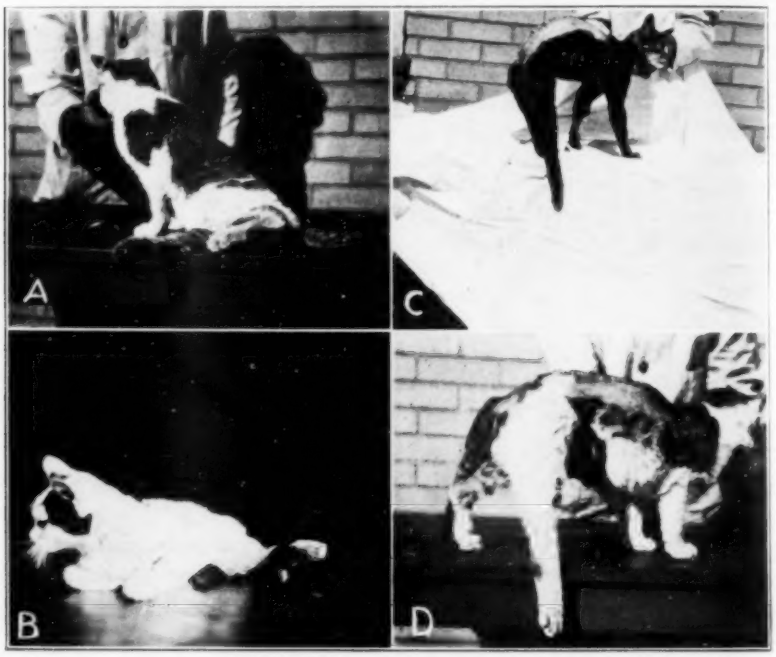

Fig. 2.—*A*, cat 60, four days after section of the dorsal roots of the right lumbosacral plexus (third lumbar to third sacral nerve, inclusive); characteristic posture, on the right hip with the right leg extended to the left. *B*, cat 7, six days after section of the dorsal roots of the right lumbosacral plexus (fourth lumbar to third sacral nerve, inclusive); right leg extended to the left. *C*, cat 61, three days after section of the dorsal roots of the right lumbosacral plexus (third lumbar to third sacral nerve); maximal extension of right leg. *D*, cat 60, four days after section of the dorsal roots of the right lumbosacral plexus (third lumbar to third sacral nerve, inclusive); maximal extension of the right leg.

which were taken from three to six days after the operation. If the cat is placed on its left side, it can rise to its feet but falls again on its right hip. For this reason the cats are always found resting on the right hip during the period of hypertonicity.

If, during the period of tonic overaction of the extensor muscles, the cat is supported by the neck, the deafferented leg is held rigidly extended and is drawn over to the left. This is illustrated in figure 3, which is a photograph of cat 13 taken eight days after section of the dorsal roots on the right side (third lumbar to third sacral, inclusive). In struggling, the cat will alternately flex and extend the other limbs, but the more violently it struggles, the more rigidly is the right leg extended. The inclination of the right leg to the left is partly due to a rotation of the pelvis and lower lumbar vertebrae. The rotation of the vertebral column is evident at autopsy, after the bones have been freed from their surrounding muscles. In addition to the rotation of the

Fig. 3.—Cat 13, eight days after section of the dorsal roots of the right lumbosacral plexus (third lumbar to third sacral nerve, inclusive); extension and adduction of the right leg.

pelvis, a hypertonia of the adductor muscles apparently is present, which helps to draw the right leg over to the left.

A good way of demonstrating this overaction of the extensor muscles in experiments, when it is not well developed, is to support the cat by the neck and tail as in figure 4 *A*. The normal leg will then be partly flexed, while the deafferented one will be fully extended and thrust somewhat backward in the posture characteristic of decerebrate rigidity

In the course of about two weeks, the cat is again able to stand and walk. A certain amount of overaction of the extensor muscles, which prevents the proper flexion of the knee and ankle, usually persists, however. The foot may not be brought forward beyond a point

directly under the hip joint, and the leg is dragged with the dorsal side of the foot down. This is illustrated in figure 4 *B*, which is a photograph of cat 1 taken fifty-seven days after section of the dorsal roots of the left lumbosacral plexus. The dragging of the leg is due not to paralysis but to the failure of the extensor muscles to relax at the proper time to allow the flexor muscles to bring the leg forward and set the foot down in the proper position.

It is easy to understand how such postures as those illustrated in figures 2 *A* and 4 *B* might lead an observer to think that the anesthetic leg was paretic. The disability, however, is due not to paralysis, but to the excessive and uncontrolled activity of the extensor muscles. No doubt the paresis of deafferented limbs described by earlier observers, Korniloff and others, was in part a pseudoparesis of this sort. In fact, Korniloff [38] stated that his dogs always held the paretic leg extended. It is, of course, possible that, with the less well developed

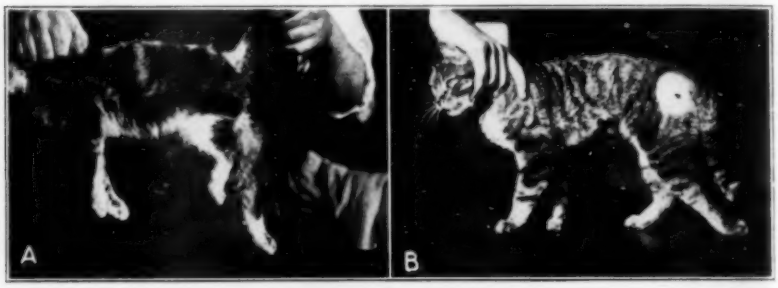

Fig. 4.—*A*, cat 13, eight days after section of the dorsal roots of the right lumbosacral plexus (third lumbar to third sacral nerve, inclusive); extension of the right leg, *B*, cat 1, fifty-seven days after section of the dorsal roots of the left lumbosacral plexus (fifth lumbar to second sacral nerve, inclusive); failure to flex the left leg which was dragged behind; the right hip had been freed from hair and the location of the femoral trochanter was marked with iodine.

technic available in those days, damage sufficient to produce a true paralysis may, in some instances, have been done to the cord.

The work of Warrington, Braeunig, Lapinsky and Trendelenburg shows that, even with the most careful technic, the exposure of the cord entails chromatolytic and degenerative changes. Such changes in the spinal cord may, of course, be responsible for the hypertonia seen in this series of experiments, but it seems more probable that they are responsible for the initial hypotonia and decreased motility which precedes the period of hypertonicity. At any rate, an overaction of the extensor muscles is regularly seen in the completely deafferented hind leg of the cat; this is in direct conflict with the usual belief that such a limb should be flaccid. The review of the literature on the

results of experimental section of the dorsal roots, in a previous section of this paper, makes it clear that previous observers have noted similar phenomena. Mott and Sherrington,[37] and Kopczynski,[48] working with monkeys, found that section of the dorsal roots of the brachial plexus caused a continuous contraction of the flexor muscles of the elbow and wrist, and the arm was continuously carried in a flexed and adducted position, as if supported by a sling. Bickel's[39] two dogs developed an extensor rigidity of the deafferented hind limb in every way similar to that seen in our cats. I believe that a large factor in the development of this overaction of the dominant muscles of the deafferented limb is the cutting off of the afferent inhibitory impulses normally coming from the limb itself. This idea has been discussed in connection with Trendelenburg's work in a preceding section of this paper.

In some cats this overaction of the extensor muscles does not develop, or, if it develops, it disappears early and the animal is able to flex

Fig. 5.—*A*, cat 4, fifty-three days after the section of the dorsal roots of the right lumbosacral plexus (fourth lumbar to third sacral nerve, inclusive); flexion of right leg with weakness of the right ankle. *B*, cat 59, five days after the section of the dorsal roots of the right lumbosacral plexus (third lumbar to third sacral nerve, inclusive); flexion of the right leg.

the leg in a nearly normal manner. In the cat illustrated in figure 5 *A*, the right leg had been completely deafferented for fifty-three days. It had never shown much stiffness and on the day when the photograph was taken the following record was made: "The right leg is weak and ataxic. In walking there is a tendency for the ankle joint to bend under the weight of the body and for the calcaneus to rest on the floor." But even in this cat, flexion was not always properly performed, as noted in the record on the same day: "Sometimes in walking the leg is not properly flexed. It is brought to a point under the hip joint and is then dragged behind with the dorsum of the foot resting on the floor until the next forward step, which may or may not involve proper flexion of the right leg." Figure 5 *B* is a photograph of cat 59, five days after section of the third lumbar to third sacral dorsal roots on the right side. This is another cat which did not at any time show

much overaction of the extensors of the deafferented leg. Such cats have been in the minority in my experiments. They are able to stand and walk within a few days after the operation because they are free from the excessive extension and adduction of the anesthetic limb, which disables the majority of the cats for many days.

In most experiments, within two or three days after the operation, more or less resistance to passive flexion of the deafferented leg developed. In four of the twelve experiments, summarized in table 1, there was not at any time resistance to passive flexion; in two others, the resistance was slight though definite; in the remaining six, resistance to flexion was marked and persisted when the deafferented leg was completely at rest. Cat 2, in which the dorsal roots had been cut on the right side from the fourth lumbar to the third sacral nerve, inclusive, developed a high grade rigidity. Seven days after the operation this cat

TABLE 1.—*Extensor Functions After Section of the Dorsal Roots*

Cat	Roots Cut	Overaction of Extensors	Resistance to Flexion	Crossed Extensor Reflex	Hyperesthesia	Artery
58	Fourth lumbar to third sacral	+	0	+	+	Cut
59	Third lumbar to third sacral	+	0	+	0	Cut
60	Third lumbar to third sacral	++	++	+	+	Damaged
61	Third lumbar to third sacral	+	+	+	+	Intact
62	Third lumbar to third sacral	+	0	+	++	Cut
63	Third lumbar to third sacral	++	++	+	++	Cut
64	Third lumbar to third sacral	++	++	+	+	Intact
65	Third lumbar to third sacral	++	+	+	+	Cut
66	Third lumbar to third sacral	++	++	+	+	Cut
67	Third lumbar to third sacral	+	0	+	0	Intact
68	Third lumbar to third sacral	++	++	+	+	Intact
69	Third lumbar to third sacral	++	++	+	+	Damaged

was suspended in a towel, as shown in figure 6 *A*. When the cat was resting quietly in this position, the right leg remained fully extended and offered considerable resistance to passive flexion. An upward pressure of 15 ounces (0.45 Kg.) against the right foot (exerted through and measured by a postal scale) was required to flex this deafferented leg. After a few minutes of repeated passive flexion and extension, the leg became relatively pliable and could be flexed by a pressure of 8 ounces (0.2 Kg.). Two hours later, the cat was again suspended in the towel and a pressure of 14 ounces (0.4 Kg.) was required to flex the leg. This slow return of rigidity after the extensor muscles were stretched was noted in a few other cats. The extensor muscles in this cat were clearly in the state of contracture; this subject will be dealt with further in another section of this paper.

When this cat was put on its back, the right hind leg projected rigidly into the air as is shown in figure 6 *B*, which reproduces a photograph taken seventeen days later than that shown in figure 6 *A*, and twenty-four days after the operation. At this time the leg still showed

high grade rigidity. It is unusual for the resistance to passive flexion to persist so long; usually it disappears within two or three weeks after the operation.

The account in the preceding paragraphs is based, for the most part, on the records of experiments conducted during the winter of 1925 to 1926. In the autumn of 1926, another series of cats was operated on in a similar manner in order to check the observations made during the earlier experiments. The results obtained were the same in the two groups. The photographs shown in figures 2 *A*, *C* and *D* and 5 *B* and the data in the three tables were obtained from the second series.

Kineographs of some of the cats of the second group were shown at the Forty-Third Session of the American Association of Anatomists in Nashville. Reproductions from this film are shown in figure 7. The great advantage of the motion pictures is the evidence which they furnish of the steady, persistent character of the positions and attitudes assumed. Contractions are being dealt with which to all outward

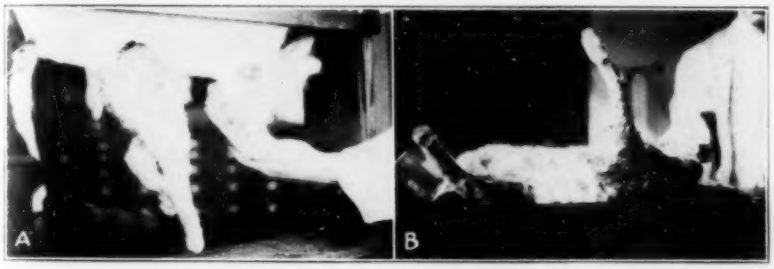

Fig. 6.—*A*, cat 2, seven days after section of the dorsal roots of the right lumbosacral plexus (fourth lumbar to third sacral nerve, inclusive); extension of the right leg. *B*, cat 2, twenty-four days after section of the dorsal roots of the right lumbosacral plexus (fourth lumbar to third sacral nerve, inclusive); extension of the right leg.

appearances are tonic, and which impose on the limb characteristic and more or less persistent postures.

Cat 60 occupies a considerable part of the cinema film, but was not included in the sections reproduced in figure 7. Two photographs of this cat are reproduced in figure 2 *A* and *D*. Figure 2 *A* shows the characteristic posture of these cats during the first ten days or two weeks after the operation. They sit on the right hip with the right leg directed forward and to the left. In figure 2 *D* the cat is shown at one side of the table with the right leg projecting over the edge. This is an excellent way to show the overaction of the extensor muscles, perhaps because of the slight traction which is thus produced on the opposite adductor muscles. In the cinema record shown in figure 7 *A* and *C*, cat 64 occupies a similar position. Another way to get the same result is shown in figure 7 *D* in which cat 64 is supported by the neck

Fig. 7.—Reproduction from motion picture film to illustrate the fully extended posture in which the deafferented leg is held. *A*, *C* and *D* represent cat 64, and *F* represents cat 63, in both of which the dorsal roots of the third lumbar to the third sacral nerve had been cut close to the cord. *B* and *E* represent a normal cat included for comparison.

and the normal leg, while the deafferented leg is rigidly held in full extension. Here again we have a stretching of the adductors of the normal leg. Magnus [50] has shown that stretching the opposite adductors calls forth an extensor reflex in his decerebellate dogs. Figure 7 *F* represents cat 63 whose right leg was so stiff that it projected rigidly into the air when the cat was resting on its back.

The crossed extension reflex could be elicited with ease and regularity in the deafferented hind limb by pinching the toes of the opposite foot. These tests were carried out with the cat resting on its side. In the normal cat, pinching the toes of one foot does not cause a regular or predictable response in the opposite hind leg. In my cats, however, such a stimulus to the normal hind foot regularly caused the deafferented leg to stiffen out in full extension at the knee and ankle, with the toes projecting in line with the leg and somewhat spread apart. Often, though not always, the claws were also protruded. This posture of full extension was usually maintained as long as the stimulus was continued, although in some cats, in which this reflex was less dominant, it would after a time give place to alternate flexion and extension.

The crossed extensor response in the deafferented right leg could be obtained in most characteristic form when the cat was lying on its right side. When the cat was lying quietly on the right side, the deafferented right leg would take up a more fully extended position and offer greater resistance to passive flexion than when the cat was lying on its left side. In some cats no difference could be noted when the animal was turned from one side to the other. In most of the experiments, however, the crossed extension reflex, the degree of extension of the resting limb and the resistance to passive flexion of the deafferented right leg were greater when the animal was lying on the right side. I have seen the same thing in decerebrate cats and dogs: when these animals lie on one side the limbs on that side are, as a rule, stiffer than those on the opposite side. Conceivably, this may be either a labyrinthine reflex or the result of stimulation of the exteroreceptors of that side by the weight of the body resting against the table. I have not attempted to determine more accurately the source of the afferent impulses involved.

Hyperesthesia of the opposite hind leg usually developed in from twenty-four to forty-eight hours after unilateral section of the dorsal roots. Although there does not seem to be a binding relation between the hyperesthesia of one leg and the overaction of the extensors in the other, the two phenomena were associated in most experiments; this fact must be taken into consideration in the interpretation of the

50. Magnus, R.: Personal communication to the author, 1926.

results (table 1). Perhaps the extensor rigidity of the deafferented limb may be interpreted as an exaggerated crossed extension reflex, occurring in the absence of the inhibitory afferent impulses, which normally come from the active muscles and which are prevented from reaching the spinal cord by the lesion in the dorsal roots.

In the twelve cats, the records of which are summarized in table 1, note was made of the artery which supplies a large part of the blood for the dorsal surface of the lumbar enlargement. It usually accompanies the fourth or fifth lumbar dorsal root of either the right or the left side. In six of the twelve cats it was located on the right side and was cut; in four it was on the left and remained intact; in two others there were two arteries, one on either side, and only one of these was cut—the blood supply was damaged to that extent. But, as table 1 clearly indicates, there is not any relation between the amount of damage to the blood supply and the stiffness or other results of dorsal root section.

In three cats the dorsal roots of the lumbosacral plexus were resected and all of the intradural part of the roots removed. The spinal ganglion was left in place with about 2 mm. of the dorsal root proximal to the ganglion. The results in these three experiments were in all respects similar to those in the experiments in which the roots were cut close to the cord.

Trendelenburg [36] found that no matter how carefully he performed his operations section of a large number of dorsal roots entailed some degeneration in the anterior and lateral columns. I have made Marchi preparations of the spinal cord in two of my cats, 67 and 69, and some scattering degeneration in the anterior and lateral columns was seen in both. The degeneration was most marked in the region of the dorsal spinocerebellar tract on the side of the operation. This is in keeping with the extensive chromatolysis seen in the cells of Clarke's column by Lapinsky.[35] The degeneration was similar and of approximately the same extent in both cats (fig. 8). But, while cat 69 showed the typical overaction of the extensor muscles and increased resistance to passive flexion, cat 67 showed only a little overaction of the extensor muscles and no increased resistance to passive flexion. It is also to be noticed that, while the degeneration is bilateral, the spasticity is always confined to the deafferented side. Obviously, the degeneration revealed by the Marchi method was not the determining factor in causing the rigidity.

EFFECT OF REMOVAL OF THE SPINAL GANGLIA

The conception of the antidromic conduction of tonic impulses, originally suggested by Trzecieski [28] and again fifteen years later by Frank [51] necessarily implies a close structural relationship between

51. Frank (footnote 7, first part).

sensory nerve fibers and a majority of the fibers of skeletal muscle. A hypothetic representation of such a relationship is given in figure 1. The possibility of axon reflex tonus and of hypertonia due to an irritation of the peripheral sensory neurons is implicit in the conception of antidromic conduction of tonic impulses. That this is not without precedent will be seen when it is remembered that the antidromic vasodilation of Bayliss [52] is mediated through the same fibers as the axon reflex vasodilation of Bruce,[53] and that inflammation of the spinal ganglion causes herpetic erythema and vesiculation of the skin. Many factors suggest the possibility that a similar mechanism is involved in muscle tonus (Ranson [27]).

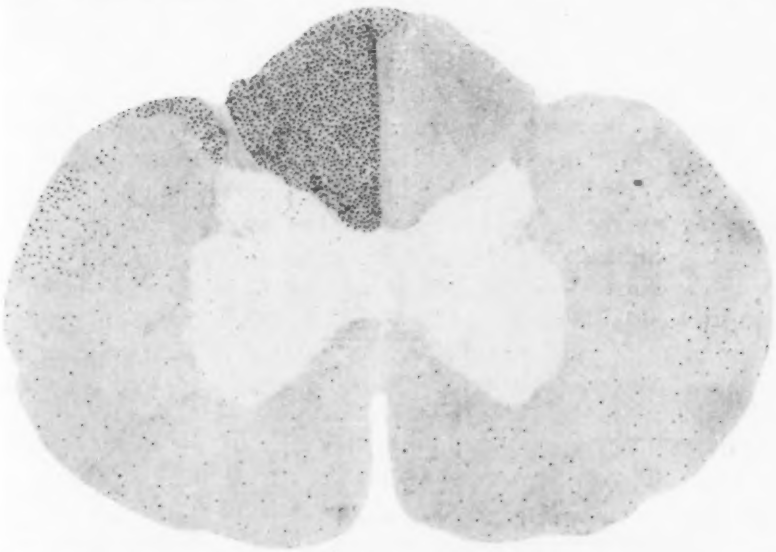

Fig. 8.—From a Marchi preparation of the third lumbar segment of the spinal cord of cat 67, killed twenty-two days after section of the third lumbar to third sacral dorsal roots of the right side.

In order to test this hypothesis, the spinal ganglia associated with the right lumbosacral plexus were removed. This operation is difficult, because it is hard to keep the field free from the blood. It was necessary, therefore, at first to limit the field; and only the sixth and seventh lumbar and the first, second and third dorsal roots were cut and the sixth and seventh lumbar and first sacral spinal ganglia more or less completely removed. In all, five cats were operated on in this manner, and in none of them did there develop either an overaction of the extensor muscles or an increased resistance to passive flexion. How-

52. Bayliss, William: J. Physiol. **36**:173, 1901.
53. Bruce, A. U.: Quart. J. Exper. Physiol. **6**:339, 1913.

ever, since the leg of the cat is so constructed that the quadriceps femoris practically controls extension at the ankle, as well as at the knee, and since the quadriceps is supplied by the fourth, fifth and sixth lumbar nerves, the first two of which were not disturbed, the experiments are without value beyond showing the necessity for completely deafferenting the leg, if one expects to get the exaggerated extensor responses already described.

In four cats I was successful in removing all of the ganglia associated with the right lumbosacral plexus. In three of these the fourth lumbar nerve was cut distal to the ganglion, and the motor fibers coming through that root were destroyed. Since this nerve, however, never contributes more than a small part of the fibers forming the femoral nerve (according to my impression gained from numerous dissections, not more than one tenth of the femoral is derived from the fourth lumbar nerve), the destruction of these motor fibers probably did not materially alter the result. In one cat the fourth lumbar spinal ganglion was removed without damage to the ventral root. In all four cats the fourth, fifth, sixth and seventh lumbar and the first sacral spinal ganglia were removed, and the second and third sacral dorsal roots were cut proximal to the ganglia. In three of these cats the completeness of the removal of the ganglia and the absence of injury to the ventral roots was checked at autopsy and by microscopic examination of the ventral roots and proximal parts of the nerves after staining with osmic acid. The results of this microscopic examination are as follows:

MICROSCOPIC EXAMINATION OF THE NERVES FROM WHICH THE SPINAL GANGLIA WERE REMOVED

CAT 48.—Autopsy was performed twenty-seven days after operation. In the fourth lumbar nerve, the ventral root was degenerated; there was no trace of the spinal ganglion. In the fifth lumbar nerve, the ventral root was normal; a few spinal ganglion cells and an occasional myelinated nerve fiber were seen in the end of the dorsal root left attached to the nerve. In the sixth and seventh lumbar nerves, the ventral roots were normal; no cells or fibers were left in the dorsal roots. In the first sacral nerve, the ventral root was normal; the distal part of the ganglion (about one sixth) was still attached to the nerve.

CAT 51.—Autopsy was performed twenty-three days after the operation. The fourth lumbar nerve was completely degenerated except for a few cells, representing the distal part of the spinal ganglion, and some fibers associated with them. In the fifth lumbar nerve, the ventral root was normal; a few spinal ganglion cells could be seen. In the sixth lumbar nerve, the ventral root was normal; there was no trace of the spinal ganglion. In the seventh lumbar nerve, the ventral root was intact; a small part of the ganglion was still attached to the nerve. No sections of the first sacral nerve were obtained.

CAT 54.—Autopsy was performed seventeen days after the operation. In the fourth lumbar nerve, the ventral root was normal; there was no trace of the spinal ganglion. In the fifth lumbar nerve, the ventral root was normal;

a little of the distal part of the ganglion (about one fifth) was still attached to the nerve. In the sixth lumbar nerve, the ventral root was intact; a little of the distal part of the ganglion (about one sixth) was still attached to the nerve. In the seventh lumbar nerve, the ventral root was intact; only a few cells and fibers of the spinal ganglion remained. In the first sacral nerve, the ventral root was normal; there was no trace of the spinal ganglion.

As is shown in table 2, only two of the four cats developed a crossed extension reflex. None of them showed great overaction of the extensor muscles at any time, and the little that was seen was late in appearing, being first noticed on the third, sixth, eleventh and fifteenth days, respectively, in the four experiments. None of these cats developed any

TABLE 2.—*Extensor Functions After Removal of the Spinal Ganglia*

Cat	Operation	Overaction of Extensors	Resistance to Flexion	Crossed Extensor Reflex
48	Cut fourth lumbar nerve distal to the ganglion; removed fourth, fifth, sixth and seventh lumbar and first sacral spinal ganglia; cut second and third sacral dorsal roots close to the spinal cord	None till after 6 days and then only slight	None	Present
51	Cut fourth lumbar nerve distal to the ganglion; removed fourth, fifth, sixth and seventh lumbar and first sacral spinal ganglia; cut second and third sacral dorsal roots close to the spinal cord	None till after 15 days and then only slight	None	Absent
54	Removed fourth, fifth, sixth and seventh lumbar and first sacral spinal ganglia; cut second and third sacral dorsal roots close to the spinal cord	None till after 3 days and then only slight	None	Present
55	Cut fourth lumbar nerve distal to the ganglion; removed fourth, fifth, sixth and seventh lumbar and first sacral spinal ganglia; cut second and third sacral dorsal roots close to the spinal cord	None till after 11 days and then only slight	None	Absent

resistance to passive flexion in the deafferented limb. In the absence of disabling rigidity in the deafferented leg, these animals were able to stand and walk earlier than the others. The removal of the ganglia, although it involved a longer operation, greater trauma and more hemorrhage, shortened the period of disability.

One must, of course, be careful not to place too great emphasis on the results of such experiments. The results could be interpreted in harmony with the theory, graphically represented in figure 1, on the assumption that the extensor rigidity following dorsal root section is caused by an irritation of the peripheral sensory neurons whose cells of origin are located in the spinal ganglia. There is some evidence that such neurons may be responsible for the tonic innervation of skeletal muscle (Frank [7] and Ranson [27]). But, on the other hand, there is the

possibility that in the dissecting away of the spinal ganglia some slight damage was done to the ventral roots, involving a temporary impairment of conductivity of the motor fibers. Against this assumption is the fact that there was no obvious paresis of the leg and, on microscopic examination, no degeneration of the motor fibers was seen.

The extensor rigidity, which results from section of the dorsal root, is out of harmony with the generally accepted idea that the integrity of the dorsal roots is essential for the maintenance of muscle tonus. It is equally opposed to the interpretation of tonus as a proprioceptive reflex or as a condition caused by the antidromic conduction of tonic impulses over the dorsal roots. The failure of this rigidity to develop after the removal of the spinal ganglia and the complete degeneration of the peripheral sensory fibers helps to bring the observations into agreement with the theory of antidromic conduction of tonic impulses, since it enables one to assume that the rigidity, which develops a few days after section of the dorsal roots proximal to the spinal ganglia, is caused by an irritation of the peripheral sensory neurons. Certainly, no one would want to overstress this argument. I do believe, however, that there is sufficient evidence in favor of the antidromic conduction of tonic impulses to justify a careful reexamination of the entire question of the relation of the dorsal roots to muscle tonus.

CONTRACTURE AFTER SECTION OF THE DORSAL ROOTS

By contracture I mean to designate a condition of permanent shortening in a muscle, which persists after all nerve impulses have been cut off by the section of the motor nerve or the death of the animal. Such a permanent shortening can be induced within five days by the action of tetanus toxin; and, when so induced, it is not due to fibrosis or to any change in the muscle fibers which can be recognized under the microscope (Ranson and Morris [54]). A similar condition of permanent shortening sometimes develops in the deafferented limb within a week or two after the section of the dorsal roots. Unfortunately this phase of the problem did not become clearly defined until late in the investigation and it is impossible to say to what extent the resistance to passive flexion noted in the earlier experiments was caused by contracture of the extensor muscles.

Cat 63, in which the third lumbar to third sacral dorsal roots were cut close to the spinal cord, developed considerable resistance to passive flexion of the deafferented leg two days after the operation. This increased in degree and persisted until the animal was killed twenty-one days after the operation. When the cat was killed with ether, the deafferented leg did not fully relax, showing that the extensor muscles were in a state of contracture.

54. Ranson, S. W., and Morris, A. W.: J. Comp. Neurol. **42**:99, 1926.

Cat 66, in which the third lumbar to third sacral dorsal roots were cut close to the spinal cord, developed a high grade resistance to passive flexion. Ten days after the operation the deafferented leg did not relax at all under ether anesthesia, which was sufficiently deep to cause full relaxation of the other three legs. Under these conditions it required ¾ pound (0.3 Kg.) pressure upward against the right foot to flex the leg. The pressure was exerted through and measured by a postal scale. Cats 68 and 69, deafferented in a similar manner, also developed high grade resistance to passive flexion. Five days after the operation cat 68 was put under deep ether anesthesia and, although the other three legs were fully relaxed, a pressure of 1¼ pounds (0.6 Kg.) was required to flex the deafferented leg. Four days after the operation, cat 69 was deeply anesthetized, and a pressure of 1¾ pounds (0.8 Kg.) was required to flex the deafferented limb. Certainly four and five days is too short a time for the occurrence of fibrosis in the muscles; and, though histologic examination of muscles in contracture following dorsal root section has not yet been made, I feel sure that this contracture is not caused by an overgrowth of the muscle by connective tissue.

It was mentioned in an earlier paragraph that a pressure of 15 ounces (4 Kg.) was required to flex the deafferented right leg of cat 2. After the extensor muscles had been stretched a few times by passive flexing and extending of the leg, the rigidity largely disappeared; but in two hours the stiffness returned again nearly to the original degree. Similar observations have been made in other cats. It was possible to pull out the contractured muscles to approximately their normal length, but in a short time they would return to their shortened state.

When the cats were kept alive for several weeks, a shortening of the flexor muscles of the toes with a tendency for the toes to be held in a position of plantar flexion nearly always developed. A similar observation was made by Trendelenburg [45] on the deafferented legs of his pigeons. The toes were arched, so that the bird could not set its foot down squarely on the floor, but walked on its toes.

I shall give further evidence of contracture in deafferented muscle in a subsequent section dealing with decerebrate rigidity. Surprisingly little is known about the kind of permanent shortening of muscle which is designated as contracture. Nothing is known about the histologic, physical or chemical changes responsible for the shortening; nor is it known what induces these changes. It is known, however, that such contracture may develop after section of the dorsal root, or in muscle poisoned with tetanus toxin; also, that it occurs in a muscle which is allowed to shorten as a result of the division of its tendon. Without doubt, the contracture, seen clinically as a result of the fixation of a limb for a long period in a plaster cast, is of the same nature. How do all these diverse conditions produce permanent shortening in muscles? This interesting and important problem has never been subjected to careful investigation.

ATROPHY

In some experiments in which the resistance to passive flexion of the deafferented leg has been especially great, I have noticed an obvious decrease in the size of the rigid quadriceps, as judged by palpation through the skin. After the rigidity disappeared the muscle seemed to fill out again. I have weighed the gastrocnemius muscles from eleven cats at varying periods after deafferentation and have not been able to detect any change from normal. The results are given in table 3. The weights there recorded show clearly that atrophy did not occur in the deafferented muscles, except, perhaps, as an early and transient alteration during the time of greatest rigidity. This serves as additional evidence that the motor neurons had not been damaged in these experiments.

TABLE 3.—*Length and Weight of Deafferented and Control Muscles After Section of the Dorsal Roots Close to the Spinal Cord, Completely Deafferenting the Right Gastrocnemius*

		Right Gastrocnemius		Left Gastrocnemius	
Cat	Period of Survival in Days	Length, Mm.	Weight, Gm.	Length, Mm.	Weight, Gm.
63	21	68.2	9.62	68.0	8.35
66	101	82.5	25.06	81.2	27.45
67	22	78.8	14.42	76.5	14.23
68	84	79.9	21.59	82.0	21.38
69	14	82.0	19.51	87.6	17.68
75	36	83.0	23.84	82.8	24.88
77	19	85.5	18.10	85.0	20.20
78	18	83.5	17.30	80.8	17.47
79	21	84.0	26.58	83.0	26.22
81	20	78.9	20.34	79.6	20.07
84	22	79.9	23.72	81.1	23.14

Atrophy, as a result of section of the dorsal root, has been seen in monkeys by a number of observers. Mott and Sherrington [37] found a certain amount of wasting but no alteration in color of the deafferented muscles. Kopczynski [48] saw an atrophy of the muscles of the thenar and hypothenar eminences in the deafferented arms. Hering [40] found, in his first monkey, some atrophy of the muscles of the deafferented arm. The upper arm was 7 mm. and the forearm 6 mm. less in circumference on the deafferented side than on the control side.

DECEREBRATION

I have employed the Pollock-Davis [55] method of decerebration.

Both carotids were ligated in the neck, and the basilar artery was clamped or tied through a small opening in the base of the skull. After decerebration, the cat was supported in a frame by strings passing through the ligaments and muscles on the dorsal side of the vertebral column (fig. 9). The head was

55. Pollock, L. J., and Davis, L. E.: Studies in Decerebration; An Acute Decerebrate Preparation, Arch. Neurol. & Psychiat. **12**:288 (Sept.) 1924.

supported and prevented from rotating laterally by a bar passing through the mouth. In measuring the rigidity in the hind limbs a postal scale with a capacity of 4 pounds (1.8 Kg.) was used. This was placed some distance in front of the leg to be tested, and the foot was drawn forward and placed on the scale pan. When the scale was pushed backward along the floor of the frame, some flexion of the leg necessarily resulted; and the pressure exerted by the foot on the scale formed an accurate measure of the rigidity. The highest pressure was developed at the moment that the backward movement ceased. After the scale became stationary, the upward pressure of the spring continued to flex the leg, at first rather rapidly and then more slowly; as this flexion occurred, the pressure decreased. The readings were taken at regular intervals, the first reading being taken at the moment the placing of the scale pan was completed. Curves constructed from readings of this sort taken at minute intervals are shown in figure 10.

I have been able to confirm Sherrington's statement that, if the dorsal roots of one lumbosacral plexus are first laid bare and the cat

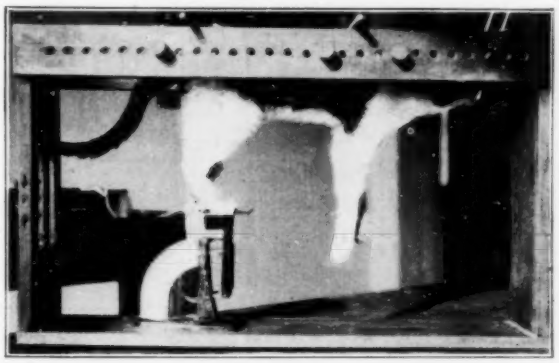

Fig. 9.—The method of stringing up decerebrate cats and the use of a postal scale in measuring decerebrate rigidity.

then decerebrated, subsequent section of the dorsal roots causes the complete disappearance of the rigidity in the corresponding hind leg. The deafferented leg becomes absolutely flaccid. But this complete disappearance of tonus immediately after dorsal root section should, I believe, be attributed in part to the shocklike depression of the motor functions of the spinal cord which results from section of the dorsal roots (Brooks [31] and Forbes and Cattell [32]).

If the dorsal roots are cut aseptically and time is allowed for recovery before decerebration is performed, a somewhat modified form of rigidity is usually present in the deafferented limb. If the decerebration is performed during the time when there is marked resistance to passive flexion, the deafferented leg shows a great deal of rigidity, although the stiffness may not be any greater after than before the decerebration. Cat 18 was decerebrated thirteen days after section of

the dorsal roots at a time when there was still a good deal of resistance to passive flexion. The stiffness of the deafferented leg persisted after decerebration, and even after the animal was killed with an overdose of ether. This is, then, another illustration of that permanent shortening of a muscle, which I have described in a preceding section under the designation of contracture. Figure 10 is a graphic record of the rigidity in the hind legs of this cat; an abbreviated protocol of the experiment follows.

Cat 18.—Feb. 9: The dorsal roots from the third lumbar to the third sacral nerve were cut on the right side.

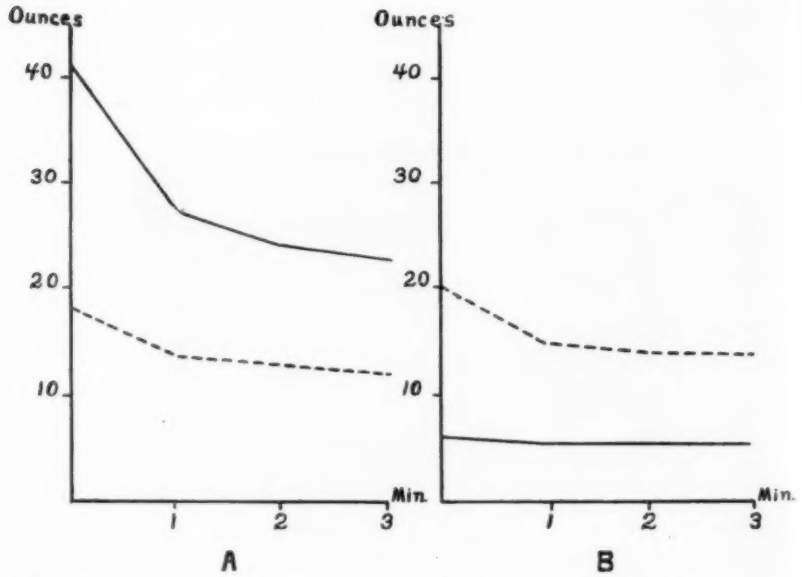

Fig. 10.—Graphic representation of the rigidity of the hind legs in cat 18 decerebrated thirteen days after section of the dorsal roots of the right lumbosacral plexus (third lumbar to third sacral nerve, inclusive). Solid line indicates normal left leg; broken line, deafferented right leg; *A*, decerebrate rigidity greater in the normal than the deafferented leg; *B*, after death, rigidity persisted in the deafferented leg but disappeared in the normal leg, 6 ounces (0.15 Kg.) representing the weight of the leg.

Feb. 22, 10:40 a. m.: There was considerable resistance to passive flexion of the deafferented right leg.

11:25 a. m.: Decerebration was completed. With the cat on its back and the head and neck symmetrically placed, the rigidity in the right hind leg was greater than in the left. The right leg remained extended in the air while the left soon became flexed at the knee. Tonic vestibular and neck reflexes were more marked in the right than in the left hind leg.

12:05 p. m.: The cat was strung up with its legs hanging downward. The right leg, though stiff, was distinctly less rigid than the left.

12:17 p. m.: The left leg registered an initial pressure of 34 ounces (0.95 Kg.) on the postal scale. The right leg registered an initial pressure of 16 ounces (0.5 Kg.).

Feb. 23, 9:10 a. m.: The cat was in good shape, with rigidity in all four legs.

12:38 p. m.: The cat was killed with ether. The left leg was entirely flaccid. The right leg remained stiff and developed an initial pressure of 12 ounces (0.3 Kg.).

12:52 p. m.: The scale was resting on a box, which raised it 1 inch (2.5 cm.) above the floor of the frame. The right leg developed an initial pressure of 20 ounces (0.6 Kg.) and the left leg an initial pressure of 6 ounces (0.15 Kg.)—the weight of the leg.

In other cats, which were kept until the resistance to passive flexion in the deafferented limb had disappeared, relatively little stiffness developed in the deafferented right leg immediately after decerebration. In periods varying in the different experiments from thirty minutes to many hours, however, this right leg became stiff. The stiff right leg did not exert a steady pressure against a hand placed beneath it, but was tremulous or even exhibited considerable oscillation under pressure. When the foot rested on the scale pan it communicated to the pan its rapid upward and downward movements, which were made more obvious because the counter-pressure exerted by the spring forced the foot upward in the intervals between the contractions of the extensor muscles.

Figure 11 represents a tracing of the upward and downward movements of the scale pan recorded in the experiment on cat 26, which was decerebrated sixteen days after section of the dorsal roots of the right lumbosacral plexus (third lumbar to third sacral nerve). The upper line represents the tracing obtained when the left foot was on the scale pan. It was taken two hours and forty minutes after decerebration. In addition to the slight oscillations there was a gradual fall in pressure during the two minutes consumed in making the record. This is easily understood when it is remembered that, as shown in figure 10, there occurs, after the scale has been placed in position, first a rapid and then a gradual fall in pressure. This tracing was made during the period of gradually falling pressure. Direct readings made from the scale while the tracing was being produced showed that the highest pressure at the beginning of the tracing was 27 ounces (0.8 Kg.) and the lowest pressure at the end was 24 ounces (0.75 Kg.). The lower tracing in figure 11 was taken immediately after the upper one, but with the right foot resting on the scale pan. It shows clearly the great and rapid fluctuations which occur in pressure. The highest pressure, as read directly from the scale while the tracing was being made, was 38 ounces (1.1 Kg.) and the lowest 17 ounces (0.5 Kg.). The decerebrate rigidity in the deafferented limb was therefore high

grade though irregular. The downward pressure exerted by the right foot was never less than 1 pound (0.5 Kg.) and at times reached more than 2 pounds (0.9 Kg.).

A slight extensor tremor develops after a few hours in most cats decerebrated by the Pollock-Davis method. The periods of heightened tonus are synchronous in all four legs and may take the form of short quick extensor thrusts. Usually, in the normal limbs, these movements are barely perceptible but can be felt by a hand placed under the foot. In a deafferented leg, however, or in one whose tonus has been

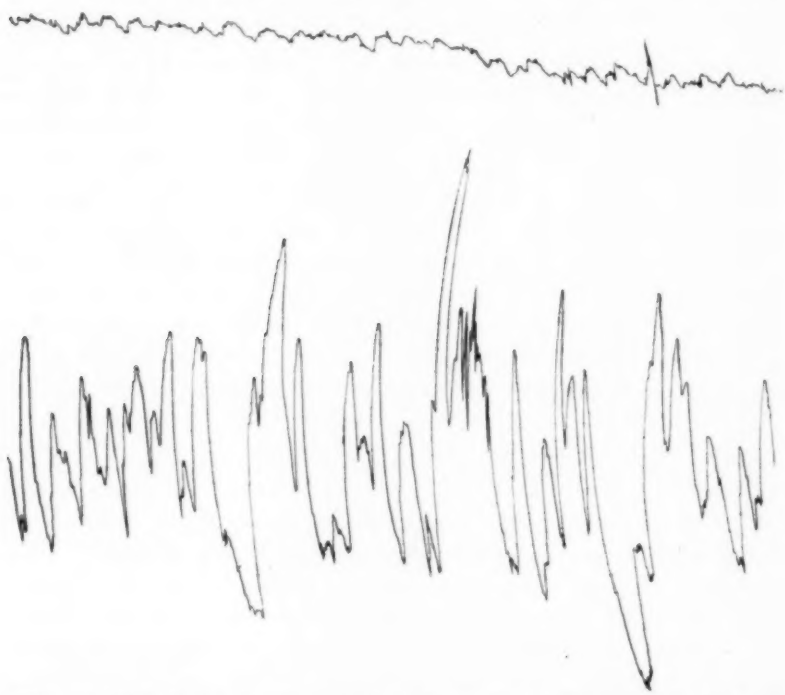

Fig. 11.—Tracings of the upward and downward movements of the scale pan—the upper tracing when the left foot rested on the pan and the lower tracing when the right foot rested on the pan—showing the great oscillations in decerebrate rigidity in the deafferented right leg; cat 26, decerebrated sixteen days after section of the dorsal roots of the right lumbosacral plexus (third lumbar to third sacral nerve, inclusive).

reduced by the application of nicotine to the lumbosacral spinal ganglia (Ranson [27]), the movements are much more extensive.

These observations are in keeping with the idea that there are two factors in decerebrate rigidity. The contractile factor is initiated by motor impulses reaching the muscle over the ventral root. It is responsible for the weak irregular action currents seen in electromyograms of muscles in decerebrate rigidity. This factor increases in prominence

with the lapse of time, after decerebration by the Pollock-Davis method, and is responsible for the slight tremor which develops in the normal limbs, and for the wide fluctuations in the downward pressure exerted by the deafferented limb. In addition, another factor confers plasticity on the decerebrate preparation and exerts a steadying influence on the contractions of the extensor muscles, dampening down the oscillations in pressure from the large fluctuations, seen in the tracing of the deafferented leg, to the barely perceptible oscillations seen in the tracing of the normal limb. Since these oscillations are synchronous in the four limbs, it is assumed that they depend on simultaneous discharges from the bulbar tonic centers. If, after section of the dorsal root, sufficient time is allowed for the disappearance of all resistance to passive flexion in the deafferented limb before decerebration is performed, this second or plastic factor is missing in the deafferented leg; an oscillating type of rigidity, different from ordinary decerebrate rigidity, develops in this leg. According to the proprioceptive reflex theory, the plastic factor is contributed by the steadying influence of the afferent impulses coming from the tonic muscles themselves. According to the theory of antidromic conduction, the plastic factor is contributed by tonic impulses, leaving the spinal cord over the dorsal roots, delaying the relaxation of the contracted muscle fibers and setting or gelling them in such a way as materially to decrease the extent of the contractions called forth by the motor nerve impulses.

In all, eight of the cats were decerebrated; and all of them—except cat 18, in which, as has been shown, the extensor muscles were in contracture—developed sooner or later this oscillating type of rigidity in the deafferented leg. In two it was evident within thirty minutes. In one cat it did not develop on the day of decerebration, but was evident the next day. One advantage of the Pollock-Davis method of decerebration is the ease with which animals are kept alive for long periods. A number of the cats were kept in good condition for more than twenty-four hours without elaborate precautions for maintaining a constant body temperature.

Tonic neck and labyrinthine reflexes, expressed as flexion or extension of the hind legs and obtained by rotating the head first to one side and then to the other or by raising or lowering the head, were easily obtained in six of the eight decerebrated cats, were, without exception, brisker on the deafferented than on the control side.

In cat 26 the extensor reflex, obtained in the hind legs by raising the head until the neck was bent sharply upward, was recorded graphically, and the tracing is reproduced in figure 12. At *A* and *B* the deafferented right foot was on the scale pan, the movements of which are recorded in the tracing. At *C* and *D* the left foot was on the pan. The letters mark the five-second periods when the head was

raised and the neck was dorsiflexed. It will be noticed that the downward pressure exerted by the right foot was unsteady; contraction and relaxation occurred in rapid succession in the extensor muscles, producing frequent and large oscillations of pressure. Toward the end of each period of stimulation a more sustained contraction occurred for a short time. When the normal left foot was on the scale, the pressure rose slowly, but steadily, and remained high through the period of stimulation. In the intervals between stimuli there were minor oscillations of pressure, which represent the tremor caused by the incompletely fused contractions of the extensor muscles and which are illustrated in figure 11.

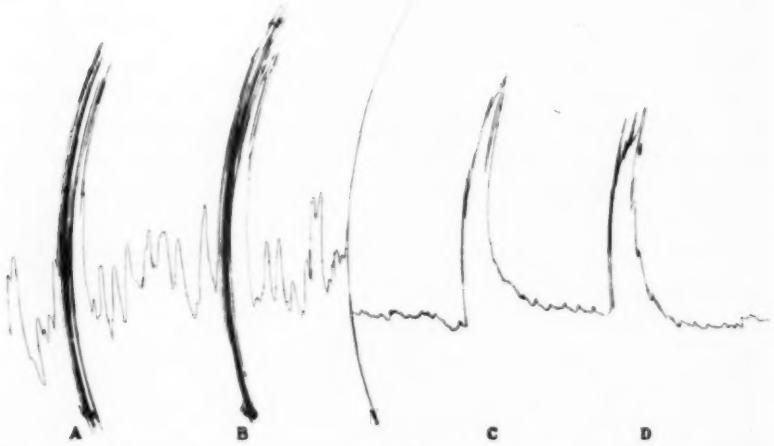

Fig. 12.—Tracings of the movements of the scale pan in the experiment on cat 26 made immediately after those shown in figure 11; *A* and *B* indicate the extensor response in the deafferented right leg elicited by raising the head and dorsiflexing the neck; *C* and *D*, extensor responses in the normal left leg elicited by raising the head and dorsiflexing the neck.

The record of cat 19 shows after decerebration that, on rotation of the head, the tonic neck and labyrinthine reflexes were quick and snappy in the deafferented right leg, while those in the left leg were slow, as if impeded by some breaking mechanism. Bickel [39] and Spiegel [56] have obtained somewhat similar results and have shown that the tonic neck and labyrinthine reflexes occur after section of the dorsal root. Spiegel argues that this invalidates the theory of the antidromic conduction of tonic impulses in the dorsal roots. But this argument will not be of much weight until it has been shown that section of the dorsal roots does not affect the steadiness and indefatigability of the tonic neck

56. Spiegel, E. A.: Arch. f. d. ges. Physiol. **193**:7, 1921.

and labyrinthine reflexes. So far as it goes, the evidence presented in figure 12 seems to indicate that the section of the dorsal roots does impair the holding power of the tonic reflexes.

The facilitation of movement in the deafferented leg was noted in other reflexes also. In cat 11, any loud sharp noise caused a sudden increase in rigidity in all four legs. In the deafferented right leg this resulted in a quick thrust of the foot downward and backward, while in the other three legs a scarcely perceptible movement occurred simultaneously.

Leaving out of consideration the experiments in which the extensor muscles were set in contracture (cat 18, fig. 10), it will be obvious that decerebrate rigidity in the deafferented leg differs from that in the normal limb chiefly in the greater mobility of the deafferented leg. In the normal leg, plasticity is highly developed; this retards and moderates all movements of the limb. The volleys of impulses from the bulbar tonic centers to the spinal cord cause simultaneous contractions of the extensor muscles in all four legs; but, while in the deafferented leg these contractions give rise to considerable oscillatory movement, in the normal leg the oscillations are impaired by the factor which makes for plasticity (fig. 11).

The contraction of the extensor muscles did not develop so quickly after decerebration on the deafferented as on the normal side. But if, after it had developed, both hind legs were flexed and held bent for a short time and then released, the deafferented right leg would snap back into full extension more quickly than the left, although the left was capable of exerting the greater pressure, as shown by the postal scale.

This plastic factor in decerebrate rigidity, which is responsible for the uniform stiffness and which causes the limb to take and keep a posture imposed on it by external force, is lost after section of the dorsal root. But this does not mean that section of the dorsal roots prevents entirely the contraction of the extensor muscles. The extensor muscles are thrown into a more or less continuous contraction, which, however, is variable and unsteady, and gives rise to an irregular and oscillating type of rigidity.

The experiments of Liljestrand and Magnus [13] point in the same direction. They found that the intramuscular injection of small doses of procaine hydrochloride greatly decreased decerebrate rigidity in the cat's triceps without completely abolishing it. These small doses did not abolish the tonic neck and labyrinthine reflexes nor the indirect excitability of the muscle. Much greater doses of procaine hydrochloride were required to abolish completely decerebrate rigidity, and these abolished at the same time the indirect excitability of the muscles. These authors found that under certain conditions they could obtain decerebrate

rigidity in a deafferented triceps; but this stiffness in the deafferented muscle was not affected by the intramuscular injection of procaine hydrochloride, except in excessive doses which also abolished the indirect excitability of the muscle.

Here again is observed the separation of the two factors in decerebrate rigidity: The contractile factor persists until the motor nerve fibers have been blocked with procaine hydrochloride. The other factor disappears when the sensory fibers have been blocked. Liljestrand and Magnus attributed this to a blocking of the sensory impulses from the muscle, and explained their results in terms of the proprioceptive reflex theory of tonus. Their results could be just as easily explained by the theory of antidromic conduction of tonic impulses along the sensory fibers which would be blocked at the same time as the sensory impulses.

SUMMARY

When, in a cat, one hind leg has been completely deafferented by section of the dorsal roots close to the spinal cord, that leg becomes atonic for only about twenty-four hours. Subsequently, an overaction of the extensor muscles nearly always develops and usually also an increased resistance to passive flexion. After two or three weeks these evidences of hypertonia largely disappear.

During the period of hypertonia the deafferented leg is adducted and extended and quite useless. Later, when the cat is again able to walk, it has difficulty in flexing this leg, which tends to be dragged with the dorsal side of the foot down. This inability to use the limb gives the appearance of a paresis.

The crossed extension reflex can be elicited with ease and regularity in the deafferented leg.

These observations agree with those of Mott and Sherrington, Kopczynski, and Bickel in showing that, after complete deafferentation of a limb by section of the dorsal roots proximal to the spinal ganglia, an overaction of the dominant muscles of that extremity may occur, resulting in fixed postures of flexion in the arm of the monkey, and extension in the leg of the cat and dog.

In four cats in which all the spinal ganglia associated with the right lumbosacral plexus were removed, the right leg never developed any resistance to passive flexion, the crossed extension reflex was difficult to elicit and such slight overaction of the extensor muscles as occurred was late in developing.

The bearing of these observations on the problem of the relation of the dorsal roots to muscle tonus is discussed, and a review made of the related literature. In spite of the great theoretical and practical importance of the problem, and notwithstanding the large amount of

work which has been done to elucidate it, the nature of muscle tonus has not been determined; nor has the rôle played by the dorsal roots in its maintenance been made clear. Two theories have been advanced in explanation of this rôle. Of these, the proprioceptive reflex theory of Sherrington is the one generally accepted; but the theory of the antidromic conduction of tonic impulses in the dorsal root, advanced by Trzecieski and Frank, has much in its favor and must be given careful consideration.

Contracture, a permanent shortening of the extensor muscles which persists after section of the motor nerves or after the death of the animal, not infrequently develops in muscles deafferented by section of the dorsal roots proximal to the spinal ganglia.

Decerebrate rigidity disappears in a leg immediately after section of its sensory fibers in the dorsal roots. This is to be attributed in part to shock. If, after section of the dorsal roots, an animal has been allowed to recover and all resistance to passive flexion has disappeared before decerebration, decerebrate rigidity is slow in developing in the deafferented limb. But after the lapse of time, varying from thirty minutes to several hours, a tremulous oscillating type of rigidity develops, owing to rapidly succeeding contractions of the extensor muscles. Tonic neck and labyrinthine reflexes occur in the deafferented limb and are usually brisker and more extensive than on the normal side.

While section of the dorsal roots does not prevent the development of decerebrate rigidity, it deprives it of one of its chief characteristics, namely, plasticity. This plasticity, which is responsible for the uniform stiffness and which causes a limb to hold a posture passively imposed on it, is dependent on the integrity of the dorsal roots. According to the theory of muscle tonus that one holds, one can explain this plasticity as a steady reflex contraction called forth by afferent impulses from the tonic muscles themselves or as due to special tonic impulses traveling antidromically over the dorsal roots.

ENCEPHALITIS PERIAXIALIS CONCENTRICA*

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The heredofamilial or endogenous diseases of the central nervous system are characterized by germ layer selectivity, system selectivity and segment selectivity (Schaffer).[1] The exogenous diseases of the brain and spinal cord, the etiology of which is unknown or which belong definitely to the group of infectious maladies, are localized in a characteristic way in the gray or white matter of the central nervous system. Both the gray and the white substance of either the brain or the spinal cord may be selectively the seat of pathologic alterations. Undoubtedly, local anatomic, physiologic and biologic factors play an important part in such selectivity. The nature of these local physiologic and biologic properties of the brain or the spinal cord is unknown. In regard to anatomic structure, it is known that the gray matter is more richly supplied with capillaries than the white substance, and this may result in physicochemical differences. In proof of the facts mentioned, it is enough to point out that epidemic encephalitis is a polio-encephalitis which is localized in the gray matter of the medulla, pons and basal ganglia. Acute anterior poliomyelitis attacks the spinal cord selectively, and in pernicious anemia the white matter is attacked without any alterations in the gray substance.

The following observation proves that the white matter of the brain may likewise be affected, without alterations in its gray substance or in the spinal cord.

REPORT OF CASE

History.—Victor P., aged 23, unmarried, a law student at the university, was admitted to the department of internal medicine of the University Hospital, Budapest, on Dec. 12, 1922. His father had committed suicide many years before; his mother, aged 44, was receiving antisyphilitic treatment. The patient's younger sister died at the age of 3 months; a second sister, aged 22, was healthy. The patient had had scarlet fever and chickenpox in infancy and influenza in 1918. He was a moderate drinker and smoker. Five years previously, he had had gonorrhea, but there was no history of syphilitic infection.

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1. Schaffer: Virchows Arch. f. path. Anat. **241**:277, 1923.

The patient said that one month before admission to the hospital, he had noticed that it became more and more difficult for him to write. He could not remember how to write certain letters. In certain words, he would stop and think how he should write the letter g, for instance. About two weeks previously, speech had become difficult and drawling, and on that account he had become taciturn and ashamed to speak. In the morning, speech was less difficult than toward evening. During the month preceding, the movements of the hands had been uncertain; for instance he had been unable to judge the position of his knife and fork at meals.

Examination.—The patient was well developed and nourished; the skin and the visible mucous membranes were slightly pale; the muscles and the bones were normal. The temperature on admission was 37.3 C. (99.7 F.). The weight was 72.6 Kg. The borders of the lungs were normal and clear throughout; dulness was not present, nor were any changes in the breath sounds heard. The heart was not enlarged; its action was regular and of good quality. The pulse rate was 92, and was rhythmic. The abdomen was normal.

The pupils were of equal size and reacted to light and in accommodation promptly. The movements of the eyes were normal and nystagmus was not present. The innervation of the right inferior branch of the facial nerve was weaker than that of the left; the pharyngeal reflex was absent. Speech was difficult and dysarthric. The patient could not write certain words or letters, though he knew them. The intelligence was normal; he was a bright student. The knee and achilles reflexes were normal on both sides. The abdominal reflexes were absent on the right. The cremasteric reflex was present on both sides. No pathologic reflexes were found. Ataxia, the Romberg sign and hypotonia were absent. Movements and reflexes of the upper extremities were normal, those of the right hand being a little awkward. Neither adiadokokinesis nor astereognosis was present. Sensation was normal. The Wassermann and Sachs-Georgi tests were negative. The fundus of both eyes was normal. Examination of the sediment of the urine showed a few red blood corpuscles and many leukocytes. The systolic blood pressure was 120, diastolic 70, as determined by Riva-Rocci's syphygmomanometer. The spinal fluid flowed with moderate pressure and was clear; the Pandy reaction was +; 3 cells were present. The patient went home on Dec. 18, 1922. He used the prescribed iodine treatment.

Second Admission.—On Feb. 21, 1923, the patient was readmitted to the hospital. His mother said that on the third day after he left the hospital the right hand and leg grew numb. Three days later, the right arm became entirely paralyzed, and the right leg was paralyzed until January 4. At the same time, the patient suffered with headaches, nausea and vomiting for about two weeks. On January 8, venesection was performed to relieve the headache. Later, quartz lamp therapy and electric treatment were carried out, in the course of which some improvement occurred, and the patient could move the paralyzed arm and leg to a limited degree. On January 18, the condition of the leg and arm again became worse, and on admission he could not use either the arm or the leg. He had been unable to sit up in bed for the past month, and his speech was disturbed. For the previous three weeks, he had not been able to speak; for the previous twelve days, he had had incontinence of urine. For the past eight days, he had suffered tonic spasms of the right arm and sometimes of the right leg also, which would last as long as half an hour. Similar cramps had been observed in the left hand and leg. He had had

trismus, and, according to his mother, the tonic cramps were induced by extreme fatigue. The cramps were preceded by profuse perspiration.

Examination.—The temperature was 37.5 C. (99.5 F.). The respiration rate was 22 and normal in type. The lungs and heart were the same as at the previous examination. The pulse was rhythmic; the rate was 108. The systolic blood pressure was 142; diastolic, 90 (Riva-Rocci's sphygmomanometer). The abdomen was normal. The patient was incontinent of urine and feces.

The pupils were round, equal, somewhat narrower than normal and they reacted promptly to light and in accommodation. Both the achilles and the knee reflexes were exaggerated, being much greater on the right than on the left. The abdominal reflex was absent in the upper and lower right segments. The cremasteric reflexes were absent on both sides. The tendon and periosteal reflexes of the right side were heightened as compared to those of the left side. On the right side. there was clonus of the tendo achillis and a positive Babinski sign. Paralysis of the facial nerve, of central origin, was present. Otherwise, the cranial nerves were normal. Nystagmus was not present. Bilateral optic neuritis was found. According to Professor Imre, both papillae nervi optici were prominent, but the blood vessels of the fundi were not engorged. In the vicinity of the disks, the retinas were clouded. There was no paralysis of the muscles of the eye, as far as could be determined while the patient was in such a disoriented condition. Tonus of the right arm and leg was increased and the muscles of the neck were spastic. The right side was totally paralyzed. There was a negative Kernig sign and a positive Trousseau sign. Sensory examination was impossible on account of the disturbed mental condition of the patient. Total motor aphasia was present, and the patient was unable to speak. He also had an almost total sensory aphasia. Detailed examination of the aphasia was impossible. The patient was capable of some movement when attempting to comply with a command and was quiet. The urine contained pus and a few red blood corpuscles and epithelial cells. The spinal fluid was clear and was under considerable pressure; the Pandy test was negative; 1 cell was present. The white blood cells numbered 9,900. On February 22, the patient had a quiet night; in the morning, the pulse rate was 134.

Because of the focal symptoms mentioned, a circumscribed alteration of the brain, especially a tumor, was considered. It was thought to involve Broca's gyrus or its vicinity. The patient was removed to the surgical department of the Metropolitan St. Stephen's Hospital. On Feb. 24, 1923, Professor Winternitz exposed the brain over an area corresponding to the left anterior central gyrus up to the sylvian fissure, and examined the Broca and Wernicke gyri. The substance of the brain did not bulge out through the opening in the skull and the structure of the gyri appeared normal. Nothing of importance was found, and the skull was closed.

On February 25, 1923, at daybreak, the patient's pulse weakened rapidly, and death occurred.

Autopsy.—At the autopsy, the internal organs were found to be normal. The histologic examination was likewise not important.

When the skull was opened, a hemorrhage was found at the temporoparietal region situated in the subdural and epidural spaces. This diffuse bleeding caused an elevation of intracranial pressure and brought about death. The brain presented moderate swelling. The gyri were somewhat flattened, and the brain substance was soft. After the removal of the brain, one section was made in the frontal plane. A small piece was excised for the purpose of animal

inoculation and another for preservation in a bromine-formaldehyde solution and a third for alcoholic fixation. Then the brain was put in formaldehyde, and after hardening was cut with the macrotome in parallel frontal planes. The first cut surface corresponded to the most anterior part of the anterior horn of the lateral ventricles (fig. 1). Here the gray matter seemed to be normal, but the white matter was altered. On cross-section, the corpus callosum was seen to be thickened, and its right half was composed of stripes of alternating white and gray substance. Each stripe was from 2 to 3 mm. in thickness. The white stripes were the same color as the white matter, while the gray stripes which were depressed, corresponded to the gray softening. The stripes extended from the gyrus cinguli in a winding manner perpendicular to the cross-section of the corpus callosum, becoming wider and forming curves up to the centrum semiovale. At about the middle of the corpus callosum these stripes presented somewhat concentric layers. On the same cut surface of the right centrum semiovale, an area 4 by 3 cm. was found. The long axis of

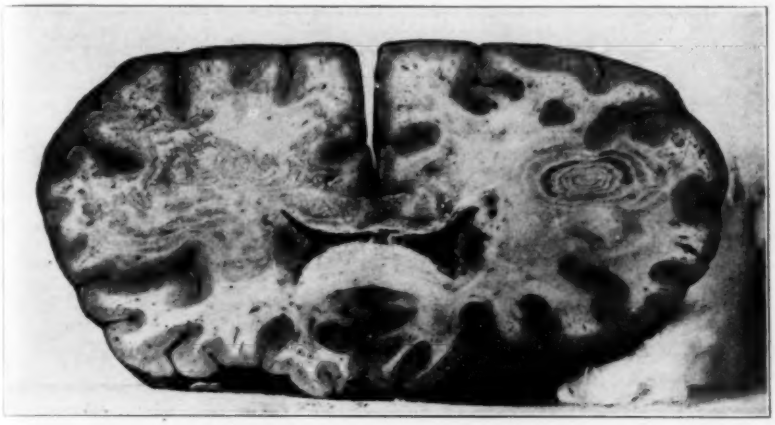

Fig. 1.—Section of the brain in the frontal plane at the anterior pole of the anterior horn of the lateral ventricles. The well defined concentric focus of alternating gray and white layers in the white substance of the right hemisphere and the scattered lines and dots in the corpus callosum and the white substance of the hemisphere should be noted.

this area was situated in the transverse diameter of the brain. The area resembled the annual rings of trees, being composed of concentric lamellae. The center of the concentric focus was an elliptic area, 3 by 1.5 mm. About this center, concentric rings composed of alternating gray and white lamellae were found. Near the center, the rings were about 1 mm. thick, while the peripheral rings were wider. It was interesting to note that where the focus approached the cortex, the rings were interrupted. The cortical substance and a thin line of white matter adjoining it were not altered. The outside rings which were the largest, penetrated even into the inferior frontal gyrus and were situated in such a way that the stripes parallel to the cortical substance were formed. The orbital gyri seemed to be normal on this side. The white matter of the left hemisphere was spotted. Gray dots the size of millet seeds and lentils were scattered throughout this region, often fusing together. In

the white substance of the inferior frontal gyrus adjacent to the gray matter, concentric stripes were to be seen running parallel to the surface. These layers resemble somewhat the peripheral part of the focus of the opposite side, though they were not as regular. On the left side, in the central part of the centrum semiovale, irregular spots were found, and no regular concentric rings had developed. While on the right side the white matter of the middle frontal gyrus was normal, the appearance of the same area of the left side corresponded to that of gray softening.

The second cut was made through the anterior commissure. The white substance of the right hemisphere appeared to be normal here. The basal ganglia, the head of the nucleus caudatus, the putamen and the globus pallidus and the internal capsule, as well as the cerebral cortex appeared normal. The apical parts of both temporal lobes found here also were normal. The axial white matter of the frontal gyri in the left hemisphere and the centrum semiovale was interspersed with gray dots. In the upper and lower frontal gyri, concentric layers were to be noticed; they ran parallel to the surface of the gyri and penetrated on the left side into the corpus callosum. These layers correspond to peripheral parts of greater concentric foci whose center was not fully developed.

The third cut corresponded to the level of the corpus mammillare. Here in the right hemisphere only the upper and middle frontal gyri contained a few winding stripes which ran parallel below the superior frontal sulcus in the form of a concentric loop from the superior frontal gyrus into the middle frontal gyrus. On the left side, the white substance of the superior, middle and inferior frontal gyri contained concentric layers which were parallel to the surface, but which were not regular. The basal ganglia, both temporal lobes, the corpus callosum and the cortical substance were normal.

The fourth cut passed through both occipital lobes at the posterior end of the lateral ventricles. In the right hemisphere a round concentric focus 2 cm. in diameter, was present and occupied the white matter of the inferior temporal gyrus external to the lateral ventricle. The rings ended as they approached the gray matter. On the right side somewhat anterior to the fissura parieto-occipitalis in the upper parietal lobe in the white substance was an elliptic concentric area, 25 by 12 mm., situated with its long axis in the transverse diameter of the brain. The peripheral rings of this ended above the posterior horn in the white substance. In the left hemisphere in the white matter of the lobulus parietalis superior and inferior, layers were found covering the upper side of the lateral ventricle, which continued in the direction of the occipital lobe.

The ganglia of the base and of the pons, medulla and spinal cord did not present pathologic alterations.

To trace the extent of one of these foci, serial sections were made to an average depth of 0.5 cm. in front of and behind the first cut surface. Behind the cut, the concentric area was localized within the white substance of the inferior frontal gyrus, and the rings became progressively larger and gradually faded away. Anteriorly, the rings become larger and less dense, but below this system of rings a new one began, which possessed a transverse diameter of 3.5 cm. and a vertical diameter of 3 cm. The second focus reached its largest distribution above the orbital gyri in the plane of the rostrum of the corpus callosum. In sections anterior to the first cut in the left hemisphere, irregular rings were found running parallel to the surface of the gyri.

Serial sections of the right occipital focus gave the same picture as that already described (figs. 2, 3 and 4). The posterior half of the occipital focus

Fig. 2.—Concentric focus of the right occipital lobe stained with Weigert medullary sheath stain. The illustration shows clearly the concentric arrangement of the degenerated white matter alternating with normal white substance. The gyri are not involved and the lesions are limited by the arcuate fibers.

Fig. 3.—A more posterior view of figure 2. The rings here are less numerous than those of the preceding figure.

was examined in Weigert preparations, while the sections of the anterior part were stained with hematoxylin-eosin. Figure 2 represents the structure of the middle portion of the occipital focus stained by Weigert's medullary sheath stain. Here the rings were most numerous. Figure 3 shows that the concentric rings were less numerous and figure 4 shows tangential sections of the focus.

All the foci showing a concentric structure possessed the form of a series of concentric globes, one fitting within the other. The gyri and sulci compressed the globes, causing irregularity of contour. In the center of a globe the concentric rings were close together, whereas they became progressively farther apart as the periphery of the focus was approached. In tangential sections, only a few irregular rings were to be found.

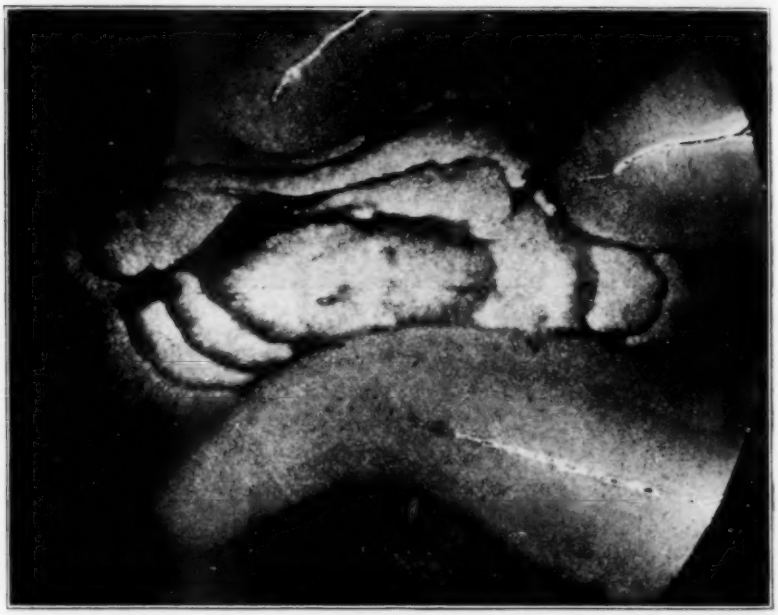

Fig. 4.—Same as figure 2, but taken still further posteriorly, showing marked diminution in the size of the concentric focus which is here cut tangentially.

In order to study the histologic structure of these concentric foci, the following staining methods were employed: (1) the medullary sheath stain of Weigert; (2) Weigert-Pal; (3) Spielmeyer's sheath stain; (4) toluidin-blue stain of Lenhossék; (5) fat stain with sudan III; (6) neuroglia impregnation according to Ramón y Cajal; (7) axis cylinder impregnation of Bielschowsky; (8) hematoxylin-eosin and van Gieson stain; (9) resorcin-fuchsin elastic fiber stain and (10) spirochete impregnation.

Histologic Examination.—The concentric foci of the brain resulted from the fact that the medullary sheaths of the nerve fibers were in alternating degenerated and intact layers. The uninjured layers of the white substance were interrupted by layers in which the medullary sheaths were destroyed. The only tissue found in the concentric foci was that of nerve fibers that had undergone degeneration. The preparations made with the Weigert, Weigert-

Pal and Spielmeyer medullary sheath stains showed best the contrast between degenerated and normal sections of nerve fibers. The rings in which the medullary sheath stain was lacking were thicker at the periphery of concentric foci, while the rings in which the medullary sheaths stained readily were found to be of the same thickness in the central and peripheral portions. The external borders of the stained concentric rings were distinct, while the internal borders gradually faded away. The concentric foci frequently reached the cortical substance, and when this occurred, they were separated from the gray matter by a thin layer of normal white matter, i. e., fibrae arcuatae. In the rings which failed to take the Weigert stain macroscopically, an occasional stained medullated fiber could be found with the microscope; these fibers were of varying thickness and were varicose, and often were broken down into

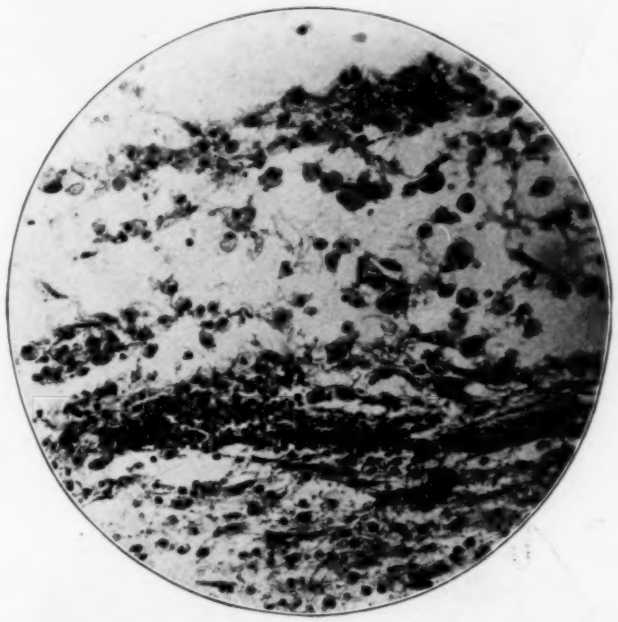

Fig. 5.—Fat granule cells distributed throughout the area of degenerated white matter with perivascular infiltration. Inferiorly, normal white substance is seen.

myelin globes. In the places in which the medullary sheath stain failed, fat droplets were present. Free fat globules occurred but the greater part was taken up by gliogenic granule cells. The sudan III stain revealed a condition that was the opposite of that shown by the medullary sheath stain. On comparing the central and peripheral rings of concentric foci it is apparent that the quantity of fat granule cells is smaller in the center than at the periphery (fig. 5). Free fat droplets seldom occurred in the central rings, and the presence of fat granule cells in perivascular lymphatic spaces was more frequent in the center than at the periphery. In other words, the breaking down of medullary sheaths seemed to be more advanced in the central rings than in the peripheral zones, and one may suppose that the destruction began in the center and progressively approached the periphery.

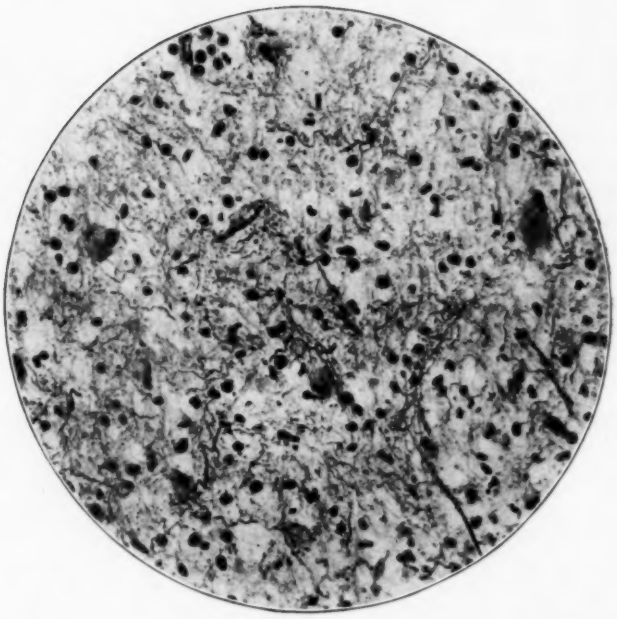

Fig. 6.—Bielschowsky impregnation of axis cylinders in the area of degenerated white matter, showing that the axis cylinders were not destroyed by the pathologic process. It is interesting to note the thickened and tortuous aspect of some of the axis cylinders.

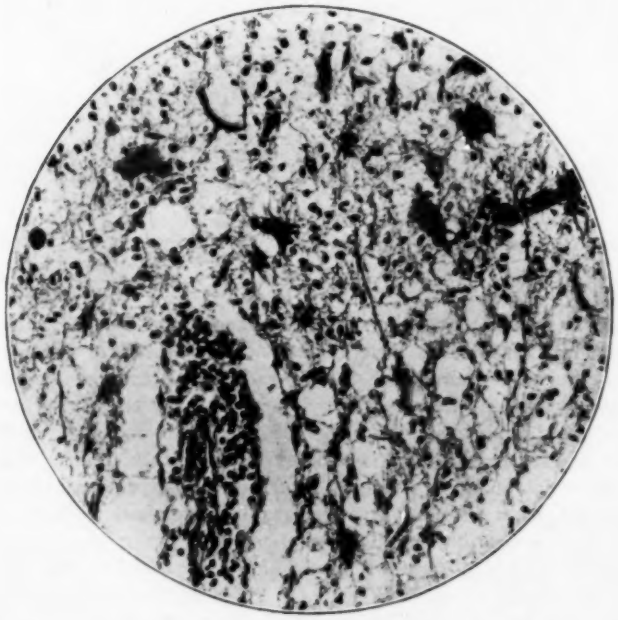

Fig. 7.—Mast glia cells in the degenerated area and perivascular infiltration.

The impregnation of the axis cylinders by Bielschowsky's stain showed that the axis cylinders of nerve fibers were destroyed in the area of concentric foci to a lesser extent than the medullary sheaths (fig. 6). In some places, however, a widespread lesion of the axis cylinders was present. The thickness of the impregnated axis cylinders varied. Thick axis cylinders were also noticed. In other places the space that occurred between the impregnated fibers was so great that one could no longer regard them as the result of the pushing away of the nerve fibers, but must suppose that a part of the fibers had been destroyed. That the greater part of the axis cylinders was preserved was proved by the fact that the medullated fibers remained in the alternating layers. According to the law of Waller, if the axis cylinders

Fig. 8.—Impregnation of the glia cells after the procedure of Ramón y Cajal. The upper portion shows normal glia cells; the lower portion, enlarged giant glia cells.

degenerated all of the fibers passing through the concentric areas should show degeneration; in the case reported, secondary degeneration was not noted.

In the layers in which the medullary sheaths were degenerated, the alteration of neuroglia was marked. It was not necessary to use special methods for staining the hypertrophic neuroglia, since all plasma stains were taken up readily by the cytoplasm and the processes of enlarged neuroglia cells (fig. 7). Progressive as well as retrogressive changes occurred in the neuroglia cells. The nuclei of proliferating glia cells were enlarged. In the faint nucleus, deeply stained nucleoli were seen. In some places, formation of marked glia fibers occurred (fig. 8). The formation of giant glia cells was most typical. The cytoplasm of such cells was either homogeneous or fine vacuoles were found at the periphery. Part of the vacuoles could be stained by fat staining dyes. Such enlarged glia cells possessed comparatively small nuclei. Some-

times small neuroglia cells surrounded these large areas. The taking up of degenerated glia cells, which is described by Schaffer,[2] Ranke[3] and Schilder[4] as gliophagia, was observed (fig. 9). The formation of neuroglia fibers was considerably greater in the center than at the periphery of the foci, again suggesting that the degeneration of the medullary sheaths began at the center.

In some parts of the brain in which no concentric foci were found but in which there were only smaller gray areas, degeneration of the medullary sheaths with intact axis cylinders was found and here there was reactive proliferation of neuroglia. Proliferation of the neuroglia occurred only in places in which the medullary sheaths were destroyed. Because of the thinness of the intact layers, giant glia cells also occurred in these layers. In the smaller dots

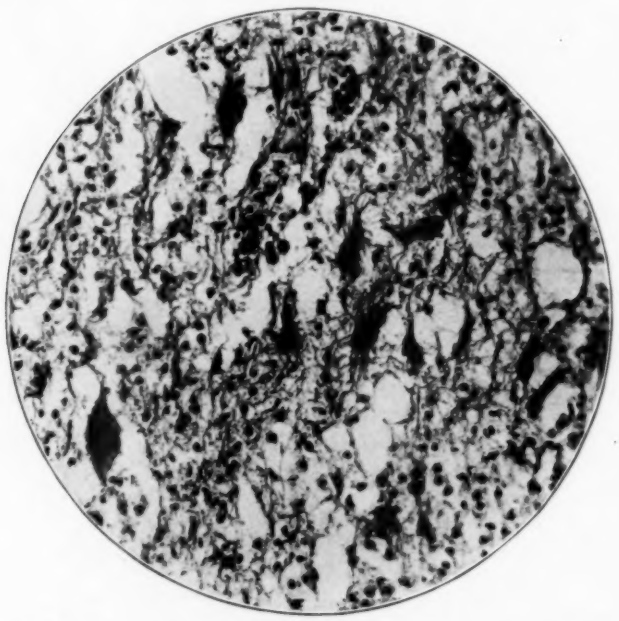

Fig. 9.—Mast glia cells of the degenerated area. Some are surrounded by small glia cells presenting gliaphagia.

which were dispersed throughout the white matter of the left hemisphere, it was evident that the proliferation of neuroglia was limited to these.

In some places in which the degeneration of the medullary sheaths was a recent process, the alteration of neuroglia was less pronounced. The reaction of neuroglia was always secondary; there was no evidence of primary tumor-like proliferation of the glia.

Though the white matter of the hemispheres presented severe pathologic alterations, the ganglia of the base, cerebral cortex, pons, medulla and cerebellum did not show changes.

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2. Schaffer: Trabajos Libro en honor de Ramón y Cajal, 1922, p. 61.
3. Ranke: Ztschr. f. d. ges. Neurol. u. Psychiat. **5**:693, 1911.
4. Schilder: Ztschr. f. d. ges. Neurol. u. Psychiat. **10**:1, 1912.

It seems impossible that the focal alterations described could be the result of alterations in the blood vessels. The size of concentric foci could not be explained by the alteration of a single medullary artery originating from the pia mater, since the foci were larger than the area nourished by a single medullary artery. In order to explain the formation of concentric foci on a vascular basis, one must suppose that several medullary arteries, running to a focus from different directions, were affected. This might result from pathologic alteration of a larger artery of the pia mater. However, if this were the case, one would expect alterations in the cortex. The intact cortex might possibly be explained on a vascular basis in that in the cortex, the blood supply is richer than in the medulla. The richer blood supply obviously must determine local physicochemical differences.

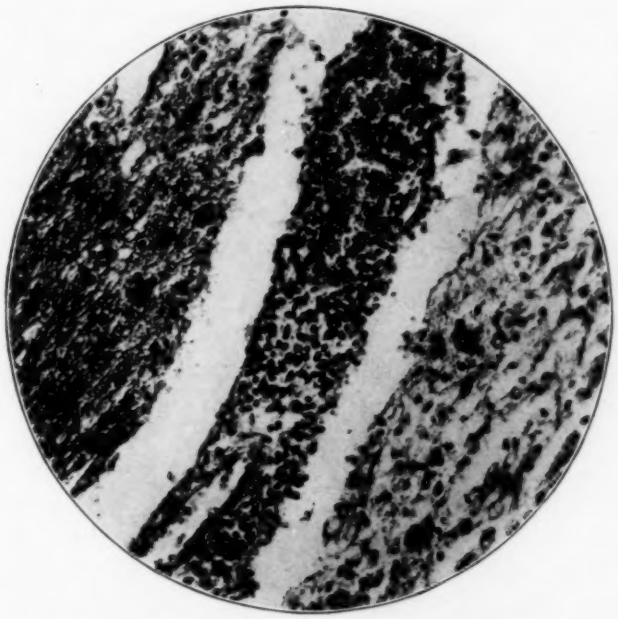

Fig. 10.—Perivascular infiltration of the degenerated area.

In areas in which the medullary sheaths were absent, the blood vessels presented perivascular infiltration (fig. 10). It has been mentioned that the periadventitial lymph spaces were filled with fat granule cells. Besides these, many lymphocytes and a few plasma cells occurred. Large mononucleated cells with a nucleus which was small when compared with the cytoplasm also were seen. Besides fat granule cells, pigment-granule cells also were found. Part of this pigment was hemosiderin. The cells described formed four or five layers around the blood vessels. The endothelium of the blood vessels was swollen, and the lumen of the small vessels was often distended with red blood corpuscles. In the brain, perivascular infiltration also occurred in the vicinity of demyelinized areas. This reactive infiltration probably also explains the alterations in the leptomeninges which were not diffuse but which were localized in that portion of the leptomeninges adjacent to a pathologic focus.

The infiltration passed along the blood vessels, through the gray substance to the leptomeninges. In the superficial layers of the basal ganglia in the vicinity of degenerated areas of white substance, slight perivascular infiltrations occurred.

Another type of lesion of the blood vessels was the hyaline thickening of the arteries (fig. 11). This was moderately pronounced and occurred also in areas separated from the foci. It was noticed in the arteries of the leptomeninges and the basal ganglia. The hyaline wall of the arteries was sometimes surrounded by perivascular infiltration. Because of the hyaline degeneration of the blood vessels, examinations for *Spirochacta pallida* were made, but gave negative results.

Fig. 11.—Hyaline thickening of the arterial walls found in both normal and diseased portions of the brain.

COMMENT

A law student, aged 23, became ill with aphasia and agraphia; then followed weakness of the inferior branch of the facial nerve and disappearance of the cremasteric reflex on both sides and of the abdominal reflexes on the right side. Later, a hemiplegia of the right side, incontinence of urine and feces and a total aphasia developed. Before death, tonic spasms occurred in the right arm and leg; trismus developed, and the patient lost consciousness. Simultaneously, the right knee reflex and the achilles reflex became exaggerated, and the Babinski phenomena appeared on the right side. At the beginning, the fundi of the eyes were normal; later, optic neuritis developed. There was moderate elevation

of temperature at this stage. The Wassermann test was negative, and a slight leukocytosis was present. Examination of the spinal fluid did not reveal any pathologic condition. Four months after the onset of the disease, an operation was performed because there were clinical manifestations of a circumscribed disease of the brain, and the left central gyrus was exposed down to the sylvian fissure. No tumor was found at operation. The patient died shortly thereafter from intracranial hemorrhage.

Postmortem examination did not reveal any pathologic conditions in the internal organs. In the white matter of the brain, foci were found varying in size from that of a lentil to that of a pigeon egg. Some resembled gray softening; others were characterized by concentric layers composed of normal white matter alternating with layers of gray softening. In the areas of gray softening fatty degeneration of the medullary sheaths was found. The fat droplets were engulfed by gliogenic granule cells. The axis cylinders of the nerve fibers in general were unchanged. When degeneration of the medullary sheaths occurred, proliferation of the neuroglia followed. The most characteristic alteration of the neuroglia was the formation of giant glia cells. These neuroglia cells showed degeneration and were the subject of gliophagia. Near the foci, the periadventitial lymphatic spaces were filled with products of degeneration, such as granule cells, lymphocytes and plasma cells. In areas of the brain widely distant from the pathologic changes just described hyaline thickenings of the arterial coats were found. The cortex, basal ganglia and spinal cord were normal.

In 1906, Marburg [5] discussed acute multiple sclerosis, basing his conclusions on his own observations and on cases described in the literature. This malady differs from the well known chronic type of multiple sclerosis because of its rapid course. The symptoms of acute multiple sclerosis are about the same as those of the chronic form. Marburg gave the name of encephalomyelitis periaxialis scleroticans to acute multiple sclerosis. According to him, acute multiple sclerosis may involve either the brain or the spinal cord separately, but generally both brain and spinal cord are coincidently attacked. Pathologically, in these cases, the alternate normal and broken down areas of medullary sheaths are characteristic, whereas the axis cylinders apparently are normal. Proliferation of the neuroglia and inflammatory alterations of the vessels complete the pathologic picture.

In 1912, Schilder [4] described encephalitis periaxialis diffusa as a new disease of the white matter of the brain, and differentiated it from the diffuse sclerosis of the brain described by Heubner,[6] who found diffuse sclerosis in the brain in a child, aged 5. This disease begins with motor

5. Marburg: Jahrb. f. Psychiat. u. Neurol. **27**:211, 1906.
6. Heubner: Charité Ann. **22**:298, 1897.

weakness; later spastic paralysis develops, and when the sclerosis becomes generalized throughout the brain, the child becomes stupid. The duration of the disease may be from a few months to perhaps one to two years. According to Heubner, diffuse sclerosis does not resemble multiple sclerosis, in which the foci are gray and are disseminated all over the brain, but is characterized by an ebony-like transformation of the white matter. Heubner's observations are based on gross examination. Schmaus [7] observed a similar case and considered it to be of inflammatory origin. In two cases mentioned, the condition occurred in the brains of children. Many authors use the term diffuse sclerosis to indicate diffuse encephalitic processes or diffuse gliomas which affect the whole brain or a whole hemisphere. The diagnosis of sclerosis of the brain has been determined in most cases by tactile examination alone.

It is to the credit of Schilder that he separated encephalitis periaxialis diffusa from the chaos of diffuse sclerosis of the brain and so created a well defined group of maladies. According to Schilder, this last disease occurs in children or in young persons and resembles tumor of the brain, multiple sclerosis or the diffuse sclerosis of Heubner, in its clinical symptoms. According to Schilder, this disease must be considered if symptoms resembling multiple sclerosis develop in young persons. The same disease has been observed in the living by Collier and Greenfield,[8] and by Claude and Lhermitte;[9] it is always fatal. Schilder said that the brains of persons who die of this disease retain the normal external appearance, but that the white matter of the hemispheres is broken down in large foci. It is possible for the white matter of the frontal, parietal, temporal or occipital lobes to be uniformly destroyed. Occasionally, the basal ganglia and the internal capsule are also involved. Recently Stewart, Greenfield and Blandy [9a] noted that the disease may affect other parts of the central nervous system, such as the pons, the optic nerves and the cervical cord. The cortical substance is in general intact and is limited by a thin layer of white matter in direct contact with it, i. e., the fibrae arcuatae. The lesion in the white matter is sharply circumscribed. Histologically, the characteristic features are: (1) the breaking down of medullary sheaths without similar alterations of the axis cylinders; (2) the appearance of large, mast glia cells, proliferation of the fibrous neuroglia and the presence of fatty granule cells; (3) infiltration of the perivascular lymphatic spaces with granule cells and lymphocytes.

Schilder's observations have been corroborated by the investigations of others. Bouman [10] mentioned perivascular infiltration of the cerebral

7. Schmaus: Virchows Arch. f. path. Anat. **114**:154, 1888.
8. Collier and Greenfield: Brain **47**:489, 1924.
9. Claude and Lhermitte: Encéphale **15**:89, 1920.
9a. Stewart, Greenfield and Blandy: Brain **50**:1, 1927.
10. Bouman: Brain **47**:453, 1924.

cortex and degeneration of the ganglionic cells of the fifth and sixth cortical layers. Similar observations have been made by Collier and Greenfield. In the case of Brock, Carroll and Stevenson,[11] the disease began in the occipital lobes and was bilateral.

In his article on acute multiple sclerosis, Marburg reported three of his own cases. In the first and second cases, both the gray and the white matter of the central nervous system were involved. In the third case, the white substance of the right hemisphere showed demyelinized areas which varied in size and which were distinctly limited in area. These were located at the right of the nucleus caudatus in the frontal lobe; but smaller foci also occurred in the white substance of the left hemisphere. The latter corresponded more or less to the picture of common multiple sclerosis. In Marburg's report, figure 10, illustrating the third case, shows that the breaking down of the medullary sheaths presented some concentricity. In this case, the cortex, basal ganglia, pons, cerebellum and spinal cord were normal. Secondary degeneration was not apparent. Ordinary multiple sclerosis affects both the white and the gray substance of the central nervous system. The gray spots of the disease occur in the brain as well as in the spinal cord. Marburg's first and second cases correspond to these pathologic changes, but his third case does not, as it shows pathologic changes in the white substance of the brain only, and in this respect it resembles the case described in this paper.

In a review of the available literature, Marburg's third case, that of Barré, Morin, Draganesco and Reys [12] and my own show the greatest similarity, but these three cases correspond much more nearly to the encephalitis periaxialis diffusa of Schilder than to acute multiple sclerosis. In these cases, only the white matter of the brain was altered. In encephalitis periaxialis diffusa, as the term points out, there is a diffuse alteration; in these three cases, however, the change was limited to circumscribed areas of the white matter of the brain. It seems probable that acute multiple sclerosis and encephalitis periaxialis diffusa are related processes.

A further question arises as to the nature of the lesions of the white matter. It is difficult to determine whether an inflammatory reaction of the central nervous system is primarily inflammatory (sensu strictiori) or a secondary reparative inflammation. In his articles on encephalitis and myelitis, Schröder [13] advised the elimination of the term inflamma-

11. Brock, S.; Carroll, P. M., and Stevenson, L.: Encephalitis Periaxialis Diffusa of Schilder, Arch. Neurol. & Psychiat. **15**:297 (March) 1926.

12. Barré; Morin; Draganesco and Reys: Rev. neurol. **33**:541, 1926.

13. Schröder: Monatschr. f. Psychiat. u. Neurol. **43**:146, 1918; Beitr. z. path. Anat. u. z. allg. Pathol. **71**:1, 1923.

tion. He asserted that, according to Nissl, the foundations of pathologic anatomy would not be shaken if the term inflammation should be left out. Schröder agreed with this and classified the alterations of the central nervous system in five groups. One of these is the myelinoclastic type, i. e., the breaking down of nervous tissue followed by the action of glia cells. In order to illustrate this type, Schröder reported the case of a man, aged 25, whose symptoms corresponded to those of encephalitis periaxialis diffusa. In the same report, Schröder referred also to a similar disease in *Cercopithecus fuliginosus*. In his classification of this group (the myelinoclastic type) he included acute multiple sclerosis; alterations in the spinal cord due to pernicious anemia; funicular and fascicular myelitis and the periaxial neuritis of Stransky,[14] an example of the alteration of normal and degenerating medullary sheaths already described by Gombault. In his first case in man, Schröder found widespread destruction of the white substance of the brain. In the perivascular lymphatic spaces, granule cells, lymphocytes and plasma cells occurred. Hemorrhagic areas in which groups of leukocytes had accumulated were found also. In his second case, in a monkey, the brain was free from leukocytes, lymphocytes or plasma cells, and only fat granule cells occurred. On account of the perivascular lymphocytic and plasma cell infiltration, many observers have regarded these cases heretofore as a real primary encephalitis. The degree of perivascular infiltration varies considerably. Walter[15] did not believe that the slight perivascular infiltration in this case indicates, essentially, a primary inflammation. In the first case of Schilder and also in those of Klarfeld,[16] Hermel[17] and Krabbe,[18] inflammatory alterations were not observed in the brain. In the second and third cases of Schilder[19] and also in those of Jakob,[20] Neubürger,[21] Henneberg,[22] von Stauffenberg,[23] Braun,[24] Marie and Foix,[25] Kraus and Weil,[26] and Guttman,[27] the inflammatory alterations were marked.

14. Stransky: J. f. Psychiat. u. Neurol. **5** and **6**:169, 1902-1903.
15. Walter: Monatschr. f. Psychiat. u. Neurol. **44**:87, 1918.
16. Klarfeld: Centralbl. f. d. ges. Neurol. u. Psychiat. **31**:50, 1923
17. Hermel: Deutsche Ztschr. f. Nervenh. **68** and **69**:335, 1921.
18. Krabbe: Ztschr. f. d. ges. Neurol. u. Psychiat. **20**:108, 1913.
19. Schilder: Ztschr. f. d. ges. Neurol. u. Psychiat. **15**:359, 1913; Arch. f. Psychiat. **71**:327, 1924.
20. Jakob: Ztschr. f. d. ges. Neurol. u. Psychiat. **27**:290, 1915.
21. Neubürger: Ztschr. f. d. ges. Neurol. u. Psychiat. **73**:336, 1921; Nissl's Beitr., 1921, vol. 4.
22. Henneberg: Neurol. Centralbl. **35**:652 and 984, 1916.
23. Von Stauffenberg: Ztschr. f. d. ges. Neurol. u. Psychiat. **39**:56, 1918.
24. Braun: Ztschr. f. d. ges. Neurol. u. Psychiat. **80**:310, 1923.
25. Marie and Foix: Rev. neurol. **27**:1, 1914.
26. Kraus and Weil: J. Nerv. & Ment. Dis. **62**:620, 1925.
27. Guttmann: Centralbl. f. d. ges. Neurol. u. Psychiat. **41**:1, 1925.

One should regard the presence of lymphocytes and plasma cells around the vessels as evidence of an inflammatory process. Epidemic encephalitis, of the inflammatory origin of which there is no doubt, is characterized by perivascular infiltration with lymphocytes and plasma cells. With the oxydase reaction, Häuptli [28] showed that only in cases of epidemic encephalitis of rapid fatality is it possible to prove the presence of leukocytes in the perivascular infiltrate.

In addition to perivascular infiltration, the vessels also show the hyaline thickening of the walls. The perivascular infiltrations correspond to the focal alterations of the white matter, but the hyaline thickening of the arterial walls occurs elsewhere. One must therefore suppose either that these lesions of the arteries are the result of the same toxic action which caused the focal alterations of the brain, or that they were present in the brain before the onset of the disease, and do not have a causative connection with it. A large number of poisons that bring about degeneration of the nerve fibers and simultaneously cause lesions of the blood vessels is known. Such a combination is observed in pernicious anemia. The Lichtheim [29] foci of the spinal cord are not the result of arterial lesions, but both they and the hyaline thickenings of the arterial walls are the result of the same toxins, which also injure the red blood corpuscles. Alcohol and lead injure the nerve fibers as well as the blood vessels. Similar action is attributed to pellagra.

Schilder's first case shows well how difficult it is to determine the inflammatory or noninflammatory character of the condition. Schilder himself regarded his first case as inflammatory. On the basis of Schilder's description, Neubürger believed that it was purely degenerative. Cassirer and Lewy [30] considered it as a blastoma. They described a condition which was similar to encephalitis periaxialis diffusa and which they regarded as a diffuse glioma because of the proliferation of the glia cells in the white substance of the brain. Glia fibers were formed in the center of the white matter, while the cell elements predominated at the periphery. The neuroglia cells grew through the cerebral cortex into the pia mater. In the case of Cassirer and Lewy, the growth of the glia cells into the cerebral cortex and the pia mater demonstrated the tumorous nature of the process and, hence, differentiated the condition in their case from encephalitis periaxialis diffusa. It is true that it is difficult to differentiate reactive glial proliferation from neuroglial tumors, and cases may occur in which this is impossible. Neubürger distinguished an inflammatory, degenerative and blastomatous type of diffuse sclerosis. He regarded his own cases as inflammatory

28. Häuptli: Deutsche Ztschr. f. Nervenh. **71**:1, 1921.
29. Lichtheim: Verhandl. d. Kong. f. inn. Med. **6**:84, 1887.
30. Cassirer and Lewy: Ztschr. f. d. ges. Neurol. u. Psychiat. **81**:290, 1923.

and considered the cases of Rossolimo [31] and Redlich [32] as similar to his own, while he believed the condition in Beneke's [33] case to be a diffuse glioma. That diffuse glioma and encephalitis periaxialis diffusa may present similar appearances is proved by the case of Cassirer and Lewy, but, in comparing the glioma and encephalitis periaxialis diffusa, a few characteristics that help in differentiation are found: 1. Diffuse glioma increases the volume of the brain and alters the gyral relations by causing increased intracranial pressure. In encephalitis periaxialis diffusa, symptoms of increased intracranial pressure are not found. 2. Grossly, the diffuse glioma is not distinctly limited, whereas in encephalitis periaxialis diffusa there are distinct limitations. Encephalitis periaxialis diffusa is confined strictly to the white substance, whereas a diffuse glioma infiltrates both the white and the gray substances. According to Stumpf,[34] normal nervous parenchyma may exist within the glioma. 3. Histologically irregular cells, hyperchromatic nuclei and nuclear divisions are signs of the presence of glioma. 4. In general, in diffuse glioma, the cellular elements are more prevalent than the fibrosis elements, and in the case of Cassirer and Lewy the fibrosis reached a high degree. According to Ranke, most fibers in a glioma do not originate in the glioma itself but in the normal neuroglia that is infiltrated by it. 5. Pressure of a glioma destroys nerve tissue. This may cause an accumulation of fat granule cells in the perivascular spaces, and slight reactive inflammatory changes occasionally occur. Well pronounced perivascular infiltration is more characteristic of encephalitis periaxialis diffusa. 6. A glioma may break through the perivascular limiting neuroglia membrane, whereas secondary gliosis generally results in a thickening of this membrane. 7. In encephalitis periaxialis diffusa, the axis cylinders of the nerve fibers are usually intact, although destruction may occur. In glioma, the destruction of nervous parenchyma reaches a higher degree on account of the increased and protracted pressure. Softenings, bleedings and the formation of cavities occur more frequently in gliomas.

Neubürger believed that a growing glioma does not necessarily involve the cortex of the brain, but that it may stop before the arcuate fibers are reached. Ángyán [35] described diffuse glioma growing symmetrically in both hemispheres.

With regard to the etiology of encephalitis periaxialis diffusa, Schilder suggested in his first communication that it is of exogenous inflammatory origin. Because of the lack of histologic examination, the

31. Rossolimo: Deutsche Ztschr. f. Nervenk. **11**:88, 1897.
32. Redlich: Wien. klin. Wchnschr. **26**:82, 1913.
33. Beneke: Arch. f. Kinderh. **47**:420, 1908.
34. Stumpf: Beitr. z. path. Anat. u. z. allg. Pathol. **51**:1, 1911.
35. Ángyán: Ztschr. f. d. ges. Neurol. u. Psychiat. **8**:1, 1911.

early cases do not throw light on this question. Jakob thought that infective agents play a part, and he stated that in his case tuberculous lesions of the lungs were found. Similar observations were made by Marie and Foix. Siemerling and Creutzfeld [36] observed the simultaneous occurrence of encephalitis periaxialis diffusa and Addison's disease. That Addison's disease may be accompanied by alterations of the brain was shown by Jakob,[37] who found atypical neuroglia reactions in this disease. Kahlden [38] also found alterations of the spinal cord similar to those of pernicious anemia, i.e., funicular myelitis in Addison's disease.

In the third case of Schilder and in the case of Klarfeld, influenza was noted. Jakob and Neubürger believed that the pathogenic agents reach the brain through the blood. In his third article, Schilder tried to prove that the variation of the connective tissue—neuroglia—and the proliferation of the blood vessels in the different cases indicate different types of virus. Braun searched for bacteria, but without success. In his case verrucous endocarditis was found, and scars due to infarctions were found in the kidneys. Braun's case, therefore, does not exactly correspond to encephalitis periaxialis diffusa, because this diagnosis can be made only if embolism is excluded. Syphilis is not considered as a cause of this disease. Schilder considered his first case nonsyphilitic in spite of a positive Wassermann reaction.

It is difficult to draw any definite conclusions regarding the infectious origin from the clinical picture alone. The disease is not accompanied by fever. In the case reported, the patient had fever and moderate leukocytosis. The temperature varied between 37.3 and 37.5 C., and the leukocyte count was 9,900. Specimens taken post mortem for bacteriologic examination revealed negative cultures. Levaditi staining of the specimens for the determination of spirochetes was negative, although a history of parental syphilis was obtained. As the cause of the disease could possibly be a filtrable virus, in order to determine this two rabbits were inoculated intracerebrally with substance from the brain according to the method used in the work in epidemic encephalitis. The rabbits presented no symptoms, and histologic investigation did not reveal any pathologic alterations.

Alteration of the vessels does not sufficiently explain the process, since the destruction of medullary sheaths extends more widely than the area supplied by one artery. Recently, Falkiewicz [39] examined the foci of the chronic form of multiple sclerosis in serial sections. He found that these foci were always independent of the areas nourished by a single artery.

36. Siemerling and Creutzfeld: Arch. f. Psychiat. **68**:217, 1923.
37. Jakob: Beitr. z. path. Anat. u. z. allg. Pathol. **69**:197, 1921.
38. Kahlden: Beitr. z. path. Anat. u. z. allg. Pathol. **10**:494, 1891.
39. Falkiewicz: Arb. a. d. Neurol. Inst. d. Wien. Univ. **28**:172, 1926.

Collier and Greenfield suggested that the primary lesions of the neuroglia of the centrum semiovale result in encephalitis periaxialis diffusa, since the nutrition of nerve fibers is dependent on the condition of the neuroglia. It would appear that several diseases exist in the central nervous system in which primary lesions of the neuroglia can be taken for granted. In the case reported in this paper, the alteration of the neuroglia was probably secondary to destruction of the medullary sheaths.

Marburg concluded that in the course of acute multiple sclerosis the lecithin component of the nerve fibers is the subject of degeneration. The axis cylinder is of albuminous nature and therefore resists the lecithinolytic action. According to Marburg, if the nerve of a frog is exposed at from 24 to 30 C. to steapsin, the medullary sheaths disintegrate but the axis cylinders remain unchanged. Lecithinolytic ferments may play a part in the chronic forms of multiple sclerosis as well as in the rapid destruction of medullary sheaths in encephalitis periaxialis diffusa. The varying factor seems to be merely the intensity of lecithinolytic action. Micro-organisms may produce the ferment. In late years, the search for the etiology of multiple sclerosis has turned toward the infections. The experiments of Bullock [40] and the investigations of Kuhn and Steiner [41] were aimed at this problem, but their observations have not been confirmed by others. According to Kutscher and Lohmann [42] lecithinolytic enzyme does not occur in the brain. Coriat [43] asserted that such enzymes destroy the lecithin of the medullary sheaths at a neutral or alkaline reaction only. One way for fat splitting enzymes to reach the circulation is in necrosis of the pancreatic fat, in which the fat tissues are split by the pancreatic enzyme—lipase. The breaking down of fats into fatty acids and glycerin occurs particularly in the vicinity of the pancreas, but it occurs also in more remote places: in the mesentery, in the tissue of the retroperitoneal fat, in the pleural cavity and in the subcutaneous fat.[44] There is still a question as to whether destruction of the medullary sheaths occurs in the brains of persons who have suffered from necrosis of the pancreatic fat.

In the case reported, the concentric layers indicate some lecithinolytic action. The clinical data suggest that the white matter in the vicinity of the gyrus of Broca was the primary seat. I was unable to demonstrate any known pathogenic agent or filtrable virus. There is no explanation of the fact that some layers of white matter remained intact, while others were destroyed. Some local immunity of the tissues may play a part in this phenomenon.

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40. Bullock: Lancet **2**:17, 1913.
41. Kuhn and Steiner: Med. Klin. **13**:1007, 1917.
42. Kutscher and Lohmann: Ztschr. f. physiol. Chem. **39**:313, 1903.
43. Coriat: Am. J. Physiol. **12**:353, 1905.
44. Aschoff: Handb., Spec. Teil, 1923, p. 899.

In order to classify my case according to the forms of diseases reported, acute multiple sclerosis and encephalitis periaxialis diffusa must be considered. The condition in my case differs from acute multiple sclerosis, since this disease affects both the brain and the spinal cord, involving the gray and white matter; nor does it correspond to encephalitis periaxialis diffusa, because of the presence of concentric foci instead of diffuse alterations. My case corresponds most closely with the third case of Marburg, and with that of Barré, Morin, Draganesco and Reys, and I would suggest the term leuko-encephalitis periaxialis concentrica for this group. Neubürger criticized the term "periaxialis." I find that the destruction of axis cylinders is never so extensive as that of the medullary sheaths and hence feel that the term periaxialis is defensible. The term "leuko-encephalitis" is justified because the inflammatory process is restricted to the white matter, i. e., where the white matter occurs in the greatest quantity, in the centrum semiovale and the corpus callosum. The school of Spielmeyer [45] uses the descriptive phrase "sclerotizing inflammation of the marrow of the hemisphere" for the same condition. Claude and Lhermitte reported that the term leuko-encephalitis was first applied by Rémond. He used this term in certain diseases of the brain associated with delirium. Rémond, however, merely hypothecated a leuko-encephalitis, but did not present anatomic evidence of it. Claude and Lhermitte recognized this disease in the living. Marie and Foix called it "sclérose intracérébrale centrolobaire et symétrique.

Leuko-encephalitis periaxialis concentrica means a disease in the course of which the white matter of the brain is destroyed in concentric layers in a manner that leaves the axis cylinders practically intact. This disease seems to be more nearly related to encephalitis periaxialis diffusa than to acute multiple sclerosis.

SUMMARY

I have described an elective and isolated disease of the cerebral white matter which is characterized by the occurrence of foci varying in size from that of a lentil to that of a pigeon's egg and presenting gray softening and, in a part, concentricity. In these foci, the medullary sheaths are destroyed, and the axis cylinders remain intact. Proliferation of the neuroglia follows the destruction of the medullary sheaths, and mast glia cells occur which partly undergo degeneration and become the subject of gliophagia. The concentric foci develop in such a manner that degenerated layers alternate with normal layers of white matter. The changes about the vessels indicate the inflammatory character of

45. Spielmeyer: Histopathologie des Nervensystems, Berlin, Julius Springer, 1922, p. 427.

the process. Syphilis, apparently, does not play a part in the etiology. Bacteriologic examination did not reveal any micro-organisms. Intracerebral inoculation with material from the brain into rabbits did not transmit the disease. One would infer from this that the causative agent is a lecithinolytic ferment that exercises its action from different foci at different intervals. Among the diseases of the brain known to date, the condition in the case reported resembles acute multiple sclerosis and the encephalitis periaxialis diffusa of Schilder. The disease differs from encephalitis periaxialis diffusa because of its focal character. It differs from multiple sclerosis because in that condition both the brain and the spinal cord and both the gray and the white matter are affected. The case reported resembles most the case of Barré, Morin, Draganesco and Reys and the third case of Marburg described in his work on acute multiple sclerosis. I suggest that the term leuko-encephalitis periaxialis concentrica be used to designate the pathologic conditions described in this paper.

ABSCESS OF THE BRAIN

REPORT OF TWO CASES—ONE WITH THE CLINICAL PICTURE OF EPIDEMIC ENCEPHALITIS AND THE OTHER WITH THAT OF TUMOR OF THE BRAIN *

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Changes in the brain resulting from an abscess necessarily vary with the type of the infection, its virulence and duration. The changes consist not only of a localized suppuration that has replaced some of the tissue of the brain, but also of reactive phenomena around the abscess. The contiguous areas either may remain intact or may exhibit more or less severe changes, depending on the intensity of the reactive phenomena. This is shown mainly as a capsule formation which checks or walls off the pus. As the reactive phenomena in young abscesses are less advanced, their membranes are also less complex and more favorable for histologic studies. The opportunity to carry on such studies presented itself in the two following cases, one cerebellar and the other cerebral.

REPORT OF CASES

CASE 1.—*Clinical History* (Dr. Bassoe).—A white woman, aged 52, admitted to the Presbyterian Hospital on Nov. 9, 1924, had been in good health until September 28, when she had been taken ill with a cold and pain on the left side of the face and head. These symptoms persisted, and after the third week frequent vomiting set in and lasted for about a month. Ten days before admission she became drowsy and after a few days diplopia and urinary retention set in. Soon after the onset, Dr. George Torrison examined the sinuses and found them normal.

Examination.—On admittance, the pupils and ocular movements were normal. Dr. E. B. Fowler found a slight bilateral optic neuritis. Marked general weakness was present, but localized paralysis of the extremities was not found. The knee reflexes were brisk, the right slightly more than the left; the other tendon reflexes were equal and normal. The plantar response was inconstant;

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more frequently an extensor response was obtained on the left side. The abdominal reflexes were not obtained. The patient was drowsy, confused and disoriented, and cooperated poorly in the examination.

Course.—During the first week in the hospital, it was found that turning of the head to the left frequently resulted in vomiting. On November 29 the leukocyte count was 16,650. The Wassermann test was negative with both the blood and the spinal fluid. The latter was clear; the cell count was 33, and a positive Ross-Jones test and a peculiar colloidal gold curve (2554442100) were present. The sugar content was 72 mg. per hundred cubic centimeters. The temperature during the first month only once or twice exceeded 99 F. The neck was stiff, but little headache was present. Urinary retention persisted. Two weeks later nystagmus developed which was more marked when the patient looked to the left and with the quick component to the left. The optic neuritis rapidly subsided in the right eye. Two minute hemorrhages were noted in the left disk on December 17, but on Jan. 10, 1925, both disks were described as practically normal.

Another spinal puncture was made on January 15; the Wassermann test again was negative; the cell count was 42; the Ross-Jones test was positive; the colloidal gold curve was almost "paralytic" (5545431000). During January the patient gradually improved, though she often complained of headache, vomiting and dizziness. She became able to void urine and to walk about the ward. She went home improved on Feb. 4, 1925, with a tentative diagnosis of epidemic encephalitis.

During the spring and summer, she had considerable frontal and temporal headache and dizziness. In the beginning of August, projectile vomiting began to appear and the patient returned to the hospital on August 7. During her absence from the hospital, she had lost 11 pounds (5 Kg.). A slight blurring of the disks was again present, and nystagmus was marked. Babinski's sign was obtained on the left side and ankle clonus on both sides. Considerable ataxia was present when the finger-to-nose test was performed. The abdominal reflexes were present. There was a suggestion of weakness in the lower facial muscles on the right, and the tongue was protruded to the right. The spinal fluid was under a pressure of 23 mm. of mercury. It gave a cell count of 19, a negative Wassermann reaction, a positive Ross-Jones test, and the colloidal gold curve was 5555532100; the sugar content was higher than before; namely, 89 mg. per hundred cubic centimeters. The leukocyte count was 12,800. During August, the temperature was normal, but in September there was a slight irregular fever which was explained by signs of slight bronchopneumonia. Headaches were severe at times, and the patient was often stuporous. The Babinski sign disappeared for a time. Swallowing gradually became difficult, and it was necessary to resort to nasal feeding.

The patient was seen by Dr. H. T. Patrick on October 10. He found restriction of upward movement of the eyes with weak convergence and suspected a tumor in the region of the fourth ventricle and corpora quadrigemina.

The leukocyte count rose to 19,350 on September 17, fell to 11,400 on September 22 and rose again to 30,600 on September 25; during the last three weeks of the patient's life, it did not exceed 16,000. From October 15 to October 23, the temperature stayed below 100, but on October 24 it rose rapidly and reached 105.2 F. before death on October 25.

The clinical diagnosis at the time of death was tumor in the posterior fossa. The presence of an abscess had been discussed but was not considered probable.

Necropsy Report (Dr. C. W. Apfelbach).—Only the cranial cavity was examined. The convolutions of the brain were flattened and the veins engorged with blood. The leptomeninges were normally thin. An abundance of slightly turbid cerebrospinal fluid was present. The left lobe of the cerebellum was adherent to the dura in an area, about 4 by 3 cm., along the lateral margin and the under surface of the left lobe. This lobe was slightly larger than the right; it was discolored pale yellow to pale yellowish brown, and was soft. In other respects, the brain was symmetrical in shape and consistency. In surfaces made by sectioning the cerebellum through the largest plane, there was one abscess from 7 to 8 mm. from the under surface; it measured 3.3 by 1.5 by 2.5 cm., and was filled with thick greenish pus; it had a lining from 1 to 1.5 mm. thick, which was finely granular; the inner surface of the cavity of the abscess was irregular. The left half of the cerebellum was pushed to the right, causing obstruction of the fourth ventricle. Between the large abscess and the fourth ventricle, the cerebellar tissue was firm, the normal markings were obliterated in most places and three other abscesses, 1.5, 0.7 and 0.8 cm. in their largest dimensions, were present in these surfaces. In these abscesses, also, a similar green pus was found; the membrane was grayish yellow and from 1 to 3 cm. thick. Altogether, these abscesses with the surrounding firm gray tissue involved about two thirds of the left lobe of the cerebellum. Noteworthy gross changes were not present in the bones of the skull, nor were changes noticed in the middle ears, sphenoid, ethmoid or frontal sinuses.

Bacteriology.—In smears of the pus from the abscesses, many gram-positive cocci were found. Acid-fast bacilli were not found in sections stained by the Ziehl-Nielsen method. By cultural methods, *Staphylococcus albus* was isolated and identified.

Anatomic Diagnosis.—The condition was diagnosed as multiple abscesses of the cerebellum; marked edema of the brain; localized fibrous cerebellar leptomeningitis; moderate general emaciation; moderate sclerosis of the basilar artery.

Microscopic Examination (Dr. Diamond).—Microscopic studies were made of the abscesses themselves and of the contiguous tissues. One of the abscesses (fig. 1 *A*) was encapsulated by a layer of adult fibrous tissue. Some portions of the layer were thick, while others consisted of only a few bands of collagen fibers. Among the fibers were scattered many fibroblasts and plasma cells, some of which were elongated. Blood vessels were not numerous, but as a rule were congested; some were greatly hypertrophied, and a few showed perivascular infiltration with lymphocytes and plasma cells. Between the connective tissue layer (*a*) and the inner mass of pus (*c*) was a broad zone of cellular elements (*b*), consisting mainly of plasma cells and macrophages, with occasional small blood vessels with swollen endothelium. These two layers, namely, an outer layer of connective tissue bordering on the cerebellar substance, and an inner, cellular layer, adjacent to the mass of pus, constituted the capsule. It differed in structure from the three capsule layers generally described, by Kölpin[1] and especially by Hassin,[2] in that it possessed only two distinct layers.

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1. Kölpin: Zur Symptomatologie u. path. Anat. des Hirnabscesses, Deutsche Ztschr. f. Nervenh. **25**:206, 1904.

2. Hassin, G. B.: Histopathological Studies on Brain Abscesses, M. Rec. **93**:91 (Jan. 19) 1918.

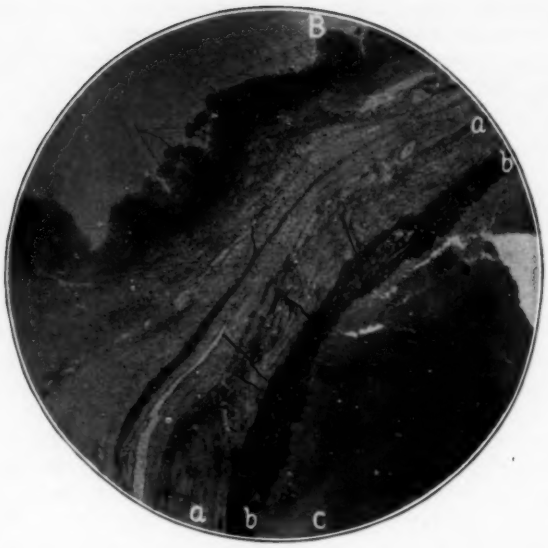

Fig. 1.—A portion of an encapsulated abscess is shown at *A* with a portion of a young pyogenic membrane of an adjacent abscess at *B*. *a* shows the outer fibrous layer; *b*, the cellular layer and *c*, pus.

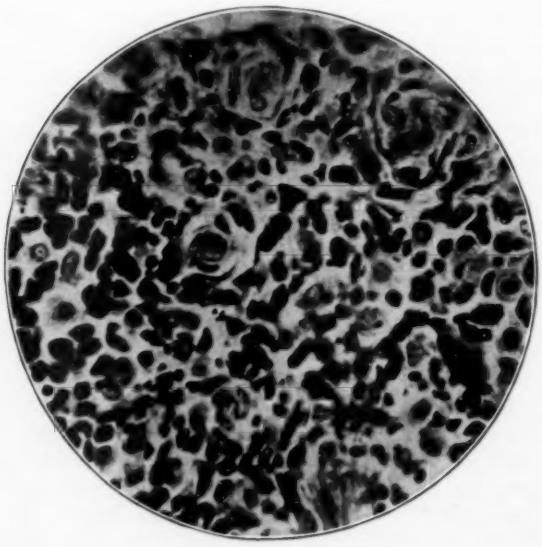

Fig. 2.—Inner layer of a young capsule in the process of formation. The blood vessels are numerous; the various cells are described in the text. Toluidin blue.

The histologic picture of the other abscesses was different. Some abscesses were small, without a definite capsule, while in others the capsules were apparently young. Instead of a well defined capsule, only a young membrane was present, which was irregular in shape and without distinct layers (fig. 1 *B*). The membrane was made up of young mesodermal elements and contained numerous blood vessels. Among these vessels were a great number of plasma cells mixed with lymphocytes, macrophages, fibroblasts, polyblasts and a few polymorphonuclear leukocytes (fig. 2). The blood vessels in the membrane were conspicuous; the endothelium was swollen and proliferated and the lumen obliterated. The same proliferative phenomena were present in the adventitial layers. The individual cells were swollen and appeared separated from each other; many were elongated or spindle shaped and resembled fibroblasts. Others were arranged in rows forming bands, and it seemed that a great part of the fibers of collagen connective tissue originated from such proliferated adventitial cells.

The smaller abscesses without definite capsules revealed changes more or less similar to those described in young membranes. In some, the cerebellar tissue was completely replaced by fibroblasts, plasma cells, lymphocytes and blood vessels; such foci did not resemble an abscess at all, for polymorphonuclear leukocytes were absent, and macrophages or gitter cells were scarce. In contrast, other foci contained numerous polymorphonuclear cells intermingled with macrophages, while at some distance from the foci they also showed a great number of plasma cells and lymphocytes. The last two types of cells were often mixed with fibroblasts which formed islands, separating the smaller necrotic foci and surrounding them, forming, as it were, young capsules.

The rest of the cerebellar tissue not occupied by these abscesses contained numerous blood vessels. They were congested; the adventitial coats were swollen and thickened and markedly infiltrated with plasma cells and lymphocytes (fig. 3). In short, the picture was one of an acute nonsuppurative encephalitis.

Noteworthy reactive phenomena could be brought out by specific methods of staining the glia, such as that of Holzer. As figure 4 shows, many islands appeared dark as compared with the surrounding cerebellar tissue. They were made up of fibers in the form of a dense network and contained a number of cytoplasmic glia cells with numerous ramifications. Such islands, generally poor in cellular elements or in blood vessels, might be classified as scars of glia tissue. In addition to the abscesses, the cerebellar tissue thus also showed foci of encephalitis and of scar formation. The remaining cerebellar tissue, however, was not altogether normal. It showed a mass of so-called glia nuclei, or oligodendroglia, some of enormous size and rich in chromatin, without signs of swelling or other regressive changes. In general, the entire left cerebellar lobe was the seat of manifold changes; they were mild in the areas remote from the abscesses but became more marked near the abscesses themselves in the form of glia scars.

Cerebral Substance: Mild changes were present in the cerebral substance at a distance from the abscesses. It was vascular, and the blood vessels were congested; some, especially the larger vessels, showed slight perivascular lymphocytic infiltrations, mainly in the optic thalami, corpora quadrigemina, substantia nigra, pons and medulla. Around some blood vessels, the tissue of the brain was somewhat rarefied; around others, clusters of glia cells were present.

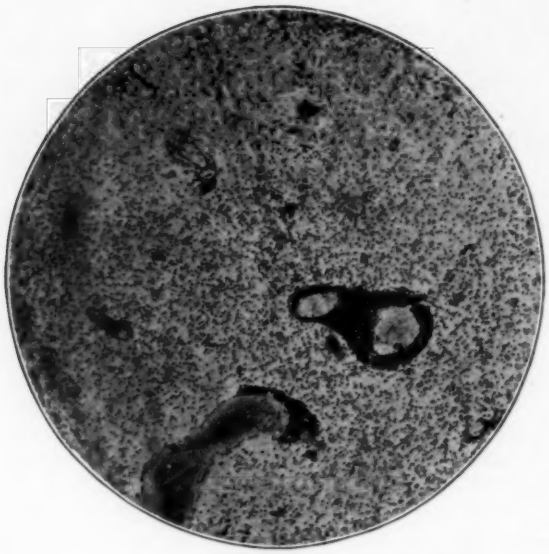

Fig. 3.—Nonsuppurative encephalitis of the cerebellar areas adjacent to the abscesses; marked perivascular infiltration. Toluidin blue.

Fig. 4.—Islands of glia scars in the cerebellum, shown in dark areas in the illustration. Holzer's stain.

The ganglion cells, as a rule, were well preserved. Some, however, exhibited degenerative phenomena — from a mild chromatolysis to complete dissolution or sclerosis. Neuronophagia and satellitosis were not uncommon. Changes in the ganglionic cells were marked in the basal ganglia and pons, while in the substantia nigra they were practically absent.

Choroid Plexus and Pia: Numerous amyloid bodies and slightly vacuolated, swollen tuft cells were present in the choroid plexus; the blood vessels were congested. The pia-arachnoid was more or less hyperplastic, especially over the frontoparietal area and over the under surface of the left cerebellar lobe. It contained well developed fibroblasts, clusters of mesothelial cells, some polyblasts, lymphocytes and polymorphonuclear leukocytes. In the region of the pineal body, cerebellum and pons, the pial meshes in addition contained gitter cells, macrophages and plasma cells. The blood vessels were congested; some showed mild perivascular infiltrations with lymphocytes, plasma cells and an occasional mast cell. Corpora amylacea were frequently seen.

Summary.—In this case were found multiple abscesses, with and without capsules, confined to the cerebellum; nonsuppurative encephalitis of the cerebellum and of the midbrain; mild degenerative changes in the ganglion cells and marked reactive leptomeningitis.

Comment.—The localization of the inflammatory phenomena in the midbrain and their absence in the hemispheres would suggest epidemic encephalitis; the encephalitic phenomena in the cerebellum were most likely secondary to the multiple cerebellar abscesses which, however, were not responsible for the encephalitis of the midbrain. The latter localization is suggestive of the epidemic type, but the abscesses cannot be ascribed to epidemic encephalitis, which is a nonsuppurative inflammation. The cause of the suppuration in this case remains unsolved. The multiplicity of the abscesses might denote a metastatic origin from the lungs or bronchi, but this could not be ascertained, as the necropsy was confined to the brain; the condition might also have been due to the weakness and incompleteness of the capsules found and their inability to protect the contiguous cerebellar tissue. Such factors were evidently at play in case 2.

Case 2.—*Clinical History.*—A woman, aged 42, was seen by Dr. L. J. Pollock, on Oct. 7, 1923, who gave us the following notes: In February, 1923, the patient became depressed, worried, imagined someone was taking her and her husband away, became self-accusatory, attempted suicide, felt that she had scandalized her husband, and presented the ordinary symptoms of a depressive psychosis, without retardation or agitation. Gradually she developed stupor with delusions of being dead and was fed through a tube for many weeks. She developed a mannerism of running her hands through her hair. On Aug. 29, 1923, she developed convulsive seizures of a generalized nature, following which she became temporarily brighter. On Sept. 4, 1923, an attack occurred in which she developed a tremor of the muscles of the face and then a localized convulsion of the face; the head and eyes turned to the left. A short time afterward, she had a generalized convulsion, after which she again

improved until September 17, when she had fever and began to vomit; this condition continued for three days. At this time, paresis of the left arm and left side of the face was noted; this gradually increased, later involving the left leg. Following this she vomited daily and complained of right-sided headache.

Examination.—Hemiplegia on the left and slight weakness of the left external rectus muscle were present. The deep reflexes were increased on the left; the left plantar reflex was not frank; the abdominal reflexes were absent; the pupils reacted to light and in accommodation. There seemed to be slight diminution of sensation over the left side, and there was a definite left homonymous hemianopia. A soft, fluctuating mass was found over the right midparietal region, and the skull under this mass was found by roentgen-ray examination to be rarefied.

A diagnosis of cerebral neoplasm, probably endothelioma, was made.

Course.—On October 23, the patient was admitted to the service of Dr. Arthur D. Bevan in the Presbyterian Hospital. It was learned that about five years previously she had struck the top of her head against a beam in a low ceiling and had complained of severe headache for some time afterward. She had never been known to have any infection of the ears or sinuses, but in the summer of 1922 she had had a carbuncle, the size of a half dollar, on the vertex and another in the left axilla. In the summer of 1923, similar lesions had been present on the upper lip and the left forearm.

Neurologic examination, Oct. 24, 1923, by Dr. Bassoe, revealed: The patient cooperated poorly on account of great mental sluggishness. She probably had left hemianopia. Hemiplegia was present on the left, including the lower part of the face. Left ankle clonus and the Babinski sign were present, while the right plantar and ankle reflexes were normal. Both knee reflexes were brisk, the left more than the right. The abdominal reflexes were not obtained. The left elbow reflex was stronger than the right. Sensation on the left side was evidently diminished but not abolished, as the patient pointed with the right hand to the place pricked with a pin. The pupils were wide and reacted to light, directly and consensually, but sluggishly. Palpable thickening of the skull was present above the right ear, which probably indicated the site of the lesion.

Ophthalmoscopic examination by Dr. T. D. Allen revealed bilateral papillitis, with a swelling of about 2 diopters.

Roentgenologic examination by Dr. C. B. Rose revealed a somewhat irregular area of bone necrosis in the right parietal region, approximately 3 by 5 cm. There was a suggestion of some thickening of the internal table in the upper anterior part of the skull vault. The sella was small but not necessarily pathologic. Other changes in the bone were not noted.

The Wassermann test with the blood was negative. The leukocyte count was 6,000; hemoglobin was 78 per cent. The systolic blood pressure was 120; diastolic, 85. The temperature did not exceed 99 F., and the pulse rate ranged from 72 to 84.

A diagnosis of probable endothelioma of the right parietal region with erosion of the skull was made, and operation was advised.

Operation.—Craniectomy was performed by Dr. Bevan on October 30. A flap with the eroded portion of the skull in the center was turned down, and the plate of bone was removed. The dura adhered to the bone by friable granulations. A piece of dura 2 inches (5 cm.) in diameter with the unhealthy adherent portion in its center was removed. No tumor presented, but when

the cortex was touched a sense of relative resistance was obtained, and the finger was pushed through the brain substance down to a firm and apparently encapsulated mass. The latter was shelled out by the finger and found to be the size of a hen's egg and apparently cystic. Pus did not issue from the wound, which was closed without drainage.

Examination of Specimens.—The piece of skull removed showed sequestration, and the cut surface showed evidence of chronic pyogenic infection. The cystic mass removed from the brain was found to be an abscess with a wall 4 mm. thick. It contained creamy, greenish yellow pus, which yielded a pure culture of staphylococcus.

Postoperative Condition.—On October 31, the temperature reached 101.4, and on November 3, 104; the pulse rate remained below 80. An irregular fever persisted, with a temperature ranging from 99 to 103 F. When some of the stitches were removed on November 6, a little bloody purulent discharge was found about them, and later there was more or less discharge of thick pus. On November 10, an aspirating needle was introduced in the direction of the former abscess, but pus was not obtained; a small amount was obtained at the upper and posterior end of the wound. For a time there was a partial return of power in the left arm and leg, and the patient was able to talk. On October 16, she had a generalized convulsion. On November 24, a note was made that the left arm was again paralyzed and that there was a fresh hemorrhage above the right optic disk, but the swelling in the disk was less than before the operation. The head was usually retracted. On December 5, difficulty in swallowing was noted, and on December 7 there were two prolonged convulsive seizures, the second one lasting fifty-five minutes and terminating in death.

Necropsy (Dr. H. A. Oberhelman).—Anatomic Diagnosis: The condition was diagnosed as localized abscess of the brain; surgical defect of the cranial bones; herniation of the brain; edema and anemia of the brain; localized multilocular abscess of the left lung; hyperplasia of the spleen; fatty changes in the kidneys, liver and myocardium; generalized emaciation and anemia and petechial hemorrhages into the lining of the stomach, duodenum and pelvic peritoneum.

The body weighed 60 pounds (27.2 Kg.) and was 153 cm. long. Emaciation was extreme. The abscess in the left lung was in the upper lobe, had a thick wall and was 3 cm. in diameter. It contained greenish-gray pus. Definite suppuration was present in the skull at the anterior angle of the surgical defect. The dura was tense and produced a definite constricting ring around the herniated portion of the brain. There was no evidence of suppuration in the leptomeninges.

Macroscopic Examination (Dr. Diamond).—The right parieto-occipital region was occupied by a protruding mass of brain tissue covered by an adherent and greatly thickened dura. This area formerly contained an encapsulated abscess that had been removed. The neighboring dura was also adherent for some distance, reaching the temporal lobe which was markedly depressed. The opposite dura could be easily stripped. The convolutions were distended and flattened. The lateral ventricle was markedly distended. A large, young abscess, the size of a small hen's egg, was situated in the right parietal lobe underneath the cortex; it was not in contact with the dura, and extended backward into the region of the cuneus of the occipital lobe. For histologic studies, sections were taken from: (1) the enucleated abscess sac (fig. 5);

Fig. 5.—Shelled out sac from the abscess of the brain; the layers are shown in figure 6.

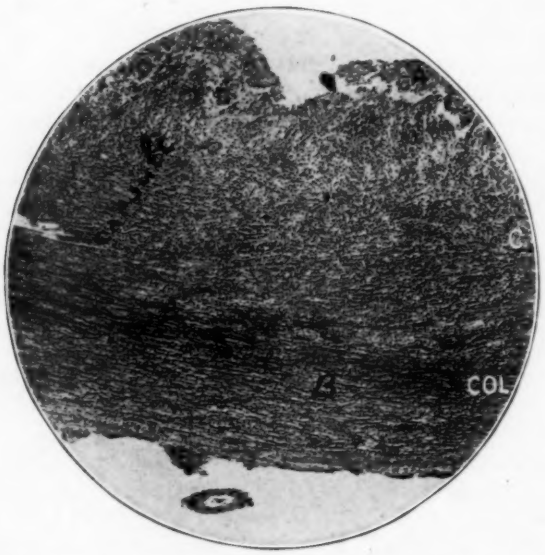

Fig. 6.—Transverse section of figure 5 (low power). *B* shows the tissue of the brain invaded by fibers of connective tissue from the outer layer of the capsule which consists of a row of fibers of dense connective tissue; *col.* shows the middle and inner layer of the capsule which are not well defined; *C*, inner layer; *A*, pus. Van Gieson stain.

(2) the pyogenic membrane of the young abscess with adjacent brain tissue from the parietal lobe; (3) the thickened dura and adjacent cortex; (4) the portion of the eroded skull flap.

Microscopic Examination (Dr. Diamond).—Transverse sections of the capsule of the abscess showed under low power a well developed layer of collagen fibers arranged in bundles (fig. 6 *Col.*). In their meshes were a number of cells, mainly fibroblasts and plasma cells, many of which were elongated. Some portion of the layer was hemorrhagic and contained numerous hyperemic blood vessels filled with polymorphonuclear leukocytes. In others,

Fig. 7.—Secondary abscess within the abscess wall, described in the text. Van Gieson stain.

the vessels were few, but their walls were thickened and the lumen was almost obliterated. Above the layer of collagen fibers was a broad zone of scattered cells, mostly macrophages, plasma cells and lymphocytes (fig. 6 *C*). The blood vessels here were minute, with swollen endothelium, and often were surrounded by fibroblasts. Collagen fibers were also present, but they were exceedingly delicate and gradually became sparse and rare near the innermost portion (fig. 6 *A*) which consisted of remnants of pus, i. e., polymorphonuclear cells and macrophages.

Outside the collagen portion of the capsule described, the conditions were somewhat complicated. As shown in figure 6, many of the collagen fibers invaded the remnants of brain tissue (*B*). The latter, however, was occupied

largely by foci of plasma cells, polymorphonuclears, macrophages, gitter cells and small congested blood vessels filled with polymorphonuclears. These cells in some places formed distinct purulent foci, surrounded by fibers of connective tissue, some of which merged with the collagen layer of the capsule. As figure 7 shows, the result was the formation of an abscess within the capsule itself.

Between the brain tissue, *B*, and the abscess, *A* (fig. 6), are reactive mesodermal phenomena in the form of a single connective tissue layer mainly

Fig. 8.—Brain tissue attached to outer layer of capsule (figures 6 and 7), showing the marked perivascular infiltration described in the text. Van Gieson stain.

bordering on the tissues of the brain. The rest of the capsule could hardly be regarded as containing separate layers, consisting mainly of numerous hematogenous elements.

The tissue of the brain attached to the outer layer of the capsule was rich in blood vessels which were markedly infiltrated with lymphocytes and plasma cells (fig. 8). Some of the vessels were bordering on the outer layer of the capsule, and their adventitial coats were greatly hypertrophied. They were surrounded by plasma cells and fibroblasts, and bands of connective tissue issued

from the outer coats of the vessels and extended for some distance into the outer capsule layer. The attached substance of the brain appeared reticular; the few remaining ganglion cells were markedly degenerated, and the glia exhibited some reactive phenomena, mainly in the form of cytoplasmic glia.

From the study of this capsule, it would seem that it was either still in the process of organization or the organization was faulty. For, notwithstanding the fact that the capsule was approximately eight months old, only the outer layer was well developed, and in addition it contained abscesses within the capsule wall, an unusual occurrence. The result was the formation of another abscess of the brain within six weeks after removal of the original one.

The recent abscess also possessed a capsule which, in structure, differed markedly from the one just described. The wall of this young abscess consisted of three layers (*A*, *B* and *C*, fig. 9), all of which were cellular and vascular.

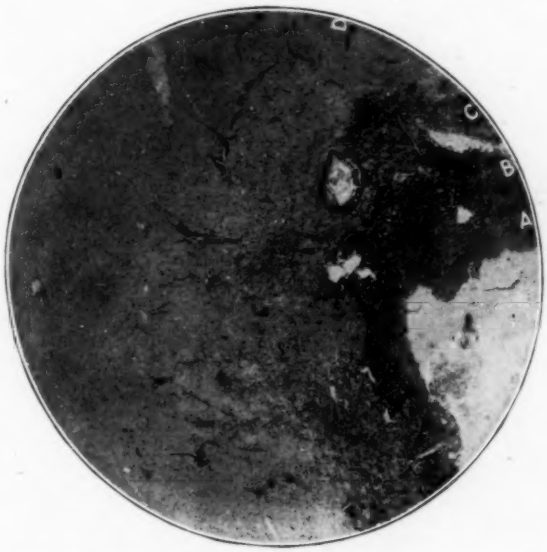

Fig. 9.—Young pyogenic membrane (six weeks old) in parietal lobe; *A*, inner layer; *B*, middle layer; *C*, outer layer; *D*, brain substance. This illustration should be compared with figure 6. Toluidin blue.

The cells were composed mostly of plasma cells, polyblasts and fibroblasts. These were especially numerous in the middle layer (*B*), while the inner and outer layers contained in addition many macrophages. Fibroblasts were especially numerous also in the middle layer and spread in all directions; they formed bands, some of which projected into the inner, some into the outer layers of the capsule. In specimens stained with van Gieson's method, delicate collagen fibers were seen projecting in all directions among the various cellular elements and young congested blood vessels. Fibers of adult connective tissue, however, were not present. Some of the larger vessels of the capsule and in the adjacent white substance were markedly infiltrated with plasma cells. The latter type of cells as well as gitter cells were also present farther away from the vessels. The glia appeared edematous, did not show reactive phenomena, and did not participate in the formation of the capsule. The contiguous ganglion cells in these sections were practically normal.

Meninges: Adjacent to the sac of the abscess, the dura and pia were adherent, greatly thickened, and converted into a membrane of fibrous connective tissue. Some portions of the membrane appeared sclerosed, some were vascular and some contained bone. A few of the blood vessels were obliterated, and many were infiltrated (plasma cells, lymphocytes), especially in the middle layers which contained many macrophages, gitter cells, fibroblasts and areas of hemorrhage. Scattered nearby were many irregular calcified and bony masses which were surrounded by osteoclasts and osteoblasts. In other words, formation of bone was going on side by side with destruction of bone. The contiguous cortex was found practically normal.

Cranial Bone: The adjacent cranial bones, of which the dura is the periosteum, were also affected (fig. 10). They showed erosions and pyogenic membranes. These consisted of young fibrous tissue and numerous blood vessels. Between the fibers were plasma cells, lymphocytes and fibroblasts.

Fig. 10.—Eroded and suppurative bone flap from the skull (parietal bone).

In some parts the cells were exclusively polymorphonuclear; others showed foci of hemorrhage. Macrophages were numerous around the latter. The blood vessels were congested, and their lumens filled with erythrocytes. In some the endothelium was swollen, prominent and proliferated. Between the pyogenic membrane and eroded bone was a well developed layer of adult fibrous bands. Many of the fibers merged with the bone, which was undergoing active absorption by a row of osteoclasts. In many portions absorption of bone was taking place within the marrow spaces, which were lined by a similar row of cells, while the marrow was being replaced by young fibrous tissue which appeared similar to that of the outer surface. In other words, the condition that we were dealing with here was a chronic osteomyelitis.

Summary.—In a patient who presented the clinical picture of a psychosis, and who had hemiplegia, two abscesses were found in the parietal lobe; one abscess was eight months old and had a fairly well defined capsule containing

a secondary abscess; the other abscess was six weeks old and had an undeveloped capsule. Marked localized pachymeningitis with calcification and ossification and osteomyelitis of the skull were present.

Comment.—Whether the clinical picture of a psychosis in this case was due to the abscess of the brain is hard to determine, though mental disorders may occur in suppurative conditions of the brain, especially when the suppurations are multiple. Reports of such cases have been collected and described by Hassin.[3] The removal of an abscess with the capsule is fraught with danger, a fact emphasized by Bagley.[4] Since the tissues of the brain are more or less adherent to the capsule, they must necessarily be torn, which may spread the infection. This is well illustrated in this case, in which such a tear was probably the cause of the secondary abscess.

GENERAL COMMENT

The two cases reported are not only of clinical but also of pathologic interest. They were especially suitable for histologic studies, mainly from the standpoint of the reactive phenomena, which could be followed in their various phases because the membranes were in various stages of development. The elements participating in the formation of the capsule were exclusively mesodermal, a fact already pointed out, especially by Hassin.[5] The contention that some capsules are of ectodermal (glial) origin was not verified by our observations. In young abscesses the protective wall is represented by masses of hematogenous elements such as lymphocytes, plasma cells, polyblasts and macrophages; they surrounded the zone of pus. In addition, the field is covered by numerous blood vessels, their walls exhibiting proliferative phenomena marked by the presence of fibroblasts. Many of these could be seen to originate from the adventitial coats of the blood vessels.

Together with other cells (lymphocytes and plasma cells), the cells (fibroblasts) ultimately change into fibrous bands forming the capsules. We may thus confirm Hassin's observations that the formation of capsules is mainly of hematogenous origin, but one must admit that the blood vessels themselves also contribute a great deal. The localized pachymeningitis in case 2 would indicate either that the infection was not entirely eradicated with the removal of the abscess sac or that a secondary infection developed after the operation.

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3. Hassin, G. B.: Dementia and Multiple Tuberculous Brain Abscesses, M. Rec., Oct. 30, 1915, vol. 88.

4. Bagley, Charles, Jr.: Brain Abscess with Pathological Observations, Surg. Gynec. Obst. **38**:1 (Jan.) 1924.

5. Hassin, G. B.: Histopathology of Brain Abscess, Arch. Neurol. & Psychiat. **3**:616 (June) 1920; footnote 2.

The presence of bone in the meninges is not uncommon in chronic inflammation with necrosis. Poscharissky [6] regarded necrosis with sclerosis as essential to calcification which in turn becomes ossified through a heteroplasia of young vascular granulation tissue surrounding the calcified masses; Leriche and Policord [7] regarded formation of bone as a metaplasia of fibrous tissue which has first been rendered embryonic. In our case the meninges in certain portions were sclerosed and contained young vascular granulation tissue which surrounded the bony and calcified masses in accordance with Poscharissky's observations. The erosions and suppurative process of the adjacent skull must be regarded as an extension from the meninges, for the outer table was found normal.

Of the numerous staining methods used, those of van Gieson and Holzer were of particular service. No particular advantages were derived from the methods of Cajal and Del Rio Hortega.

CONCLUSIONS

1. Multiple circumscribed suppurative lesions of the central nervous system may be confined to one lobe of the cerebellum and may give a clinical picture suggestive of encephalitis or of tumor of the brain with an uncertain localization.

2. Reactive phenomena are always more or less in evidence around the abscess, however young it may be, and result in the formation of a capsule.

3. The capsule is mainly formed by hematogenous elements and partly by the adventitial cells of the blood vessel walls.

4. The capsule itself may be the seat of secondary abscesses.

5. Reactive phenomena may also obtain far from the abscess, resulting either in a nonsuppurative encephalitis or in mild reaction of the glia tissue.

6. Reactive phenomena on the part of microglia and oligodendroglia were not observed.

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6. Poscharissky, J. F.: Ueber heteroplastische Knochenbildung, Beitr. z. path. Anat. u. z. allg. Pathol. **38**:135, 1905.

7. Leriche, R., and Policord, A.: Les problèmes de la physiologie normale et pathologique de l'os, abstr. in Surg. Gynec. Obst. **43**:821, 1926.

APHASIA

A STUDY OF NORMAL CONTROL CASES *

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PHILADELPHIA

Investigations of aphasia from the clinical standpoint have been confined almost entirely to the determination of the exact type of speech disorder in the various types of aphasic patients. Clinicians have been content to make use of certain tests in the examination of these patients, and have been satisfied that any deviation from the correct response has been an indication of an aphasic reaction. For general purposes this is satisfactory, in that any good series of tests for aphasia will give a general indication of the nature of the speech disorder present. For the purpose of careful inquiry into the mechanism behind aphasic disorders, however, this is not sufficient, and all the responses of an aphasic person must be carefully analyzed and evaluated. In order to do this it must be perfectly clear what constitutes an aphasic response as compared with a normal reaction; or, to put it briefly, there must be standards for comparison, so that the interpretations of results in examining various types of aphasia may be accurate. It is essential to know, therefore, exactly what constitutes a normal response, what may be looked on as a deviation from this normal, and how a normal person without demonstrable speech disorder will respond to the various tests used in the study of aphasia. If this and the errors a normal person will make are known, it is possible to decide accurately what constitutes error in an aphasic response, and to evaluate these responses properly in the study of speech. Although this seems obvious, it is extremely surprising how few studies of such a nature have been made; in attacking the problem of aphasia, we first of all set ourselves the task of determining the nature of the responses that normal persons give when the tests used in the study of aphasia are applied to them.

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* Read at the Fifty-Third Annual Meeting of the American Neurological Association, Atlantic City, N. J., May, 1927.

TYPE OF MATERIAL

The material studied consisted of twenty-nine persons. As the purpose of this study was to determine the responses of nonaphasic persons of as many types as possible to these tests, these twenty-nine persons were grouped into six classes.

Class A was composed of six graduate students in the neuropsychiatric department of the University of Pennsylvania. As these subjects were interested in the results and were conversant with psychometric examinations and tests for aphasia, they composed a group of greater intellectual keenness than the patients whom Head studied. However,

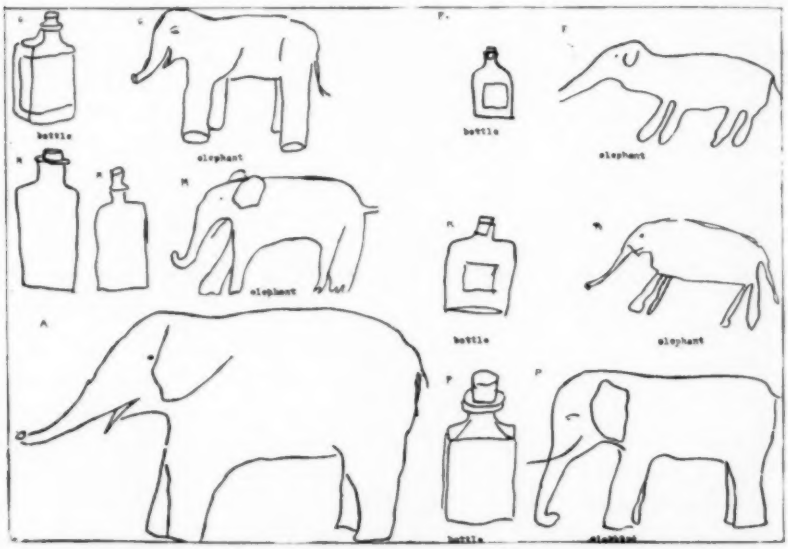

Fig. 1.—Drawings from subjects in class A.

they approach more nearly to his cases than do any of the other groups examined, for he insists that he selected intelligent persons of the higher social strata as the most fruitful in gaining a better understanding of disorders of speech.

Class B was composed of nine young adult patients in the wards of the Orthopedic Hospital; only three of this group had organic disease of the nervous system; two suffered from chronic encephalitis and one from epilepsy. All of them had reached at least the upper grades of the public school, and several had completed the first two years in high school.

Class C consisted of seven patients from the neurologic wards of the Philadelphia General Hospital who had hemiplegia of the left side.

They were elderly men whose social and intellectual levels were much inferior to those of the preceding group, but they were selected because their cases were outstanding examples of cortical disease and therefore more nearly comparable with cases of aphasia.

Class D comprised two patients with generalized cerebral arteriosclerosis. The intellectual and social status was better than that of the patients with hemiplegia, but the organic brain disorder placed them in a position analogous to that class.

Class E consisted of four patients with stationary tabes and without any signs of cerebral involvement. They were elderly men with a restricted intellectual background.

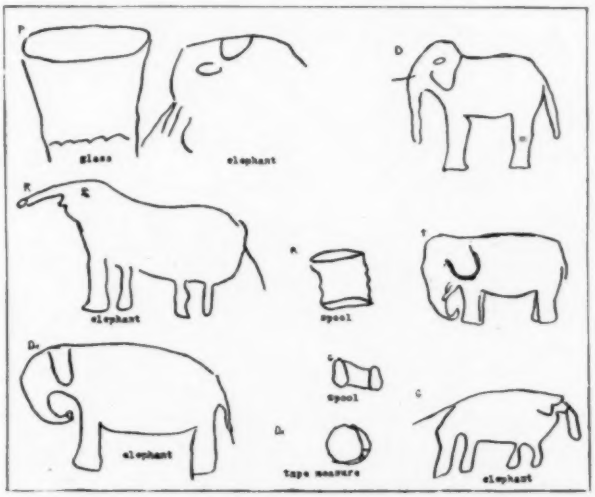

Fig. 2.—Drawings from subjects in class B.

Class F was composed of one patient with general paralysis on whom only a partial examination was made.

The control material, therefore, consisted of two main groups: persons with superior and average intelligence without organic nervous disorder, and persons with organic brain disease of different types. The subjects in both these groups were without speech disorders. In selecting the control material, an attempt was made to obtain young persons without nervous disease, in order to perceive what reactions healthy, nonaphasic persons would give. In addition, however, patients with organic brain disease but without disturbances of speech were selected in order to approach more closely the condition of the aphasic patient who has brain disease of a highly localized nature.

The tests used in the study of our patients with aphasia and in our control cases were those published by Henry Head. We were prompted to use these by a desire to determine their efficacy in the study of aphasia. The tests applied to our control material corresponded exactly to those given to patients with aphasia, and our report includes a discussion of the normal deviations which are to be expected in these tests, as well as a statement of the value of these tests in the study of aphasia.

Head does not present any systematic scheme for applying his series of tests, and a perusal of the protocols of his cases indicates that he arranges his tests differently for different patients. This method has advantages in studying individual cases, but we felt that a definite arrangement would assist in comparing the responses of different patients. Moreover, it seemed to us that by commencing the examination with the simplest tests and increasing the difficulties gradually, a better

TABLE 1.—*Type of Control Cases*

Class	Type of Case	Number	Comment
A	Superior intelligence	6	Graduate students in neuropsychiatry in University of Pennsylvania
B	Average intelligence	9	Three of these had organic brain disease
C	Left hemiplegia	7	
D	Cerebral arteriosclerosis	2	
E	Tabes dorsalis	4	Stationary cases
F	General paralysis	1	Partial examination
	Total	29	

insight into the mechanism of aphasia might be obtained. This we did, but, in order to eliminate fatigue, we alternated a difficult group of tests with one that would be less tiring. The grouping of the tests devised for aphasic patients was used as a routine in examining these control cases, with two exceptions. The record of the patient's spontaneous speech, which is of vital importance in investigating aphasia but worthless in this series, and the group of tests dealing with the patient's name and address were omitted. This left a battery of thirteen groups of tests arranged in the following order: (1) the performance of a single act in response to a simple oral command, (2) recognition of the names and values of coins, (3) recognition and naming of common objects, (4) the alphabet tests, (5) performance of complicated acts in response to oral written commands, (6) clock tests, (7) recognition and naming of colors, (8) reading and writing of simple sentences, (9) reading and writing of an easy paragraph, (10) performance of more complicated acts in response to an oral command, (11) drawing tests, (12) coin and bowl tests and (13) recognition and naming of geometric figures. A fuller description of each series will be given in presenting the results.

Although the tests were given in this order, the examination of any one patient was limited to an hour at a time, so that several days were consumed in completing a single record. During the examination, one of us gave the tests and another recorded verbatim the subject's reactions. Examiner and recorder alternated on successive days. The subjects were encouraged to take all the time they required in responding to a test, and an effort was made to make them feel at ease during the examination.

ANALYSIS OF MATERIAL

As this is an attempt to set up a standard whereby the results obtained from an aphasic patient examined by this series of tests may be

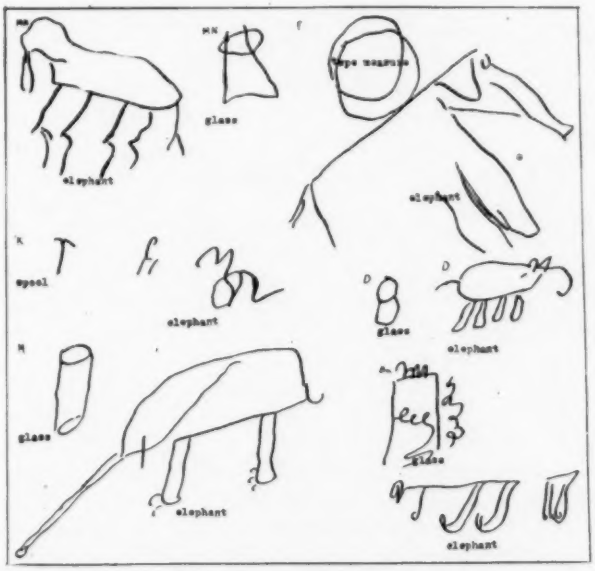

Fig. 3.—Drawings from subjects in class C.

evaluated, it seems better to discuss the responses of these control cases to each group of tests than to discuss the total reaction of any of the case groups to the whole series.

Simple Commands.—The first test consists of four parts The subject is asked to touch his nose with his right hand, then with his left hand, then his nose or his eye with his right and left hands. The first part of this test was done without error by all our subjects. There does not seem much reason for the second part of this test. The introduction of the alternative is not more confusing to aphasic patients than it is to normal persons. Several types of reactions were noted. Some subjects touched the eye only, others the nose, others touched both the eye and the nose with either hand. Less commonly the nose was touched on one side, the eye on the other. One patient in group D touched the forehead with one hand and the eye and the nose with the other.

Coin Tests.—The second group of tests was well done by all except three patients in group C. One subject without visual defect called a nickel a quarter and a penny a dime. The explanation for this is difficult as he did well in many of the harder tests. Another who had the visual defects of old age called a nickel a dime, a quarter a nickel, and, in comparing the value of a penny and a nickel, said the former was worth more.

Common Objects.—Series 3 consists of seven parts. A tray containing a knife, key, pencil, match, tape measure, scissors and a penny is placed before the patient, and he is asked to indicate on the tray the object corresponding to a duplicate shown to him. He is then asked to name the object indicated, select it from oral command, indicate it when its duplicate is placed in his hand out of sight, and write the name of the object shown. As a general rule, this series was performed faultlessly, but three subjects in group C made errors. One who was slightly deaf selected the pencil when asked to point out the penny; when the test was repeated, this error was corrected. It might be presumed that this was the result of confusion of the names due to defective hearing, but when a penny

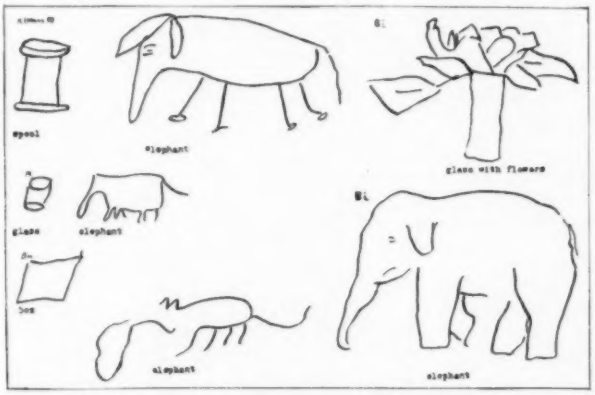

Fig. 4.—The drawings of the spool and the large elephant in the upper left corner, marked *K* (class 4) were made by subject in class D; the rest were made by subjects in class E.

was placed in his hand after his eyes were closed, he felt it, and opening his eyes, selected the pencil as the duplicate object. When handed a card asking him to point out the key, he pointed first to the pencil and then corrected his error. Another patient in the second last test indicated a knife as the duplicate of an Eversharp pencil, but on repeating the test made no mistake. The Eversharp pencil confused the third patient. In the first part of the series when shown an Eversharp pencil, the duplicate on the tray being an ordinary pencil, he hesitated a long while without making a selection. He was asked what the object shown to him was, and, on naming it correctly, he immediately selected the duplicate pencil.

Alphabet Tests.—In testing aphasic persons, it was astounding to find the number who were unable to repeat and write the alphabet from memory or even to copy it correctly. This defect was so marked that we were led to consider this series of tests of great importance in studying aphasia. When these tests are analyzed, it is perceived readily that they have little to do with speech. Oral and written repetition of the alphabet is really a test of memory of school work. Repeating it after the examiner or copying it does test the ability to recognize and

reproduce letters, but this part of the series depends a great deal on attention, as most people show the tendency to reproduce a well known succession such as the alphabet from memory rather than to follow precisely the directions for the test. Normal educated people illustrate this well, for on being asked to copy a printed alphabet they reproduce it in cursive script instead of printing the letters. Persons with a meager education, on the other hand, are inclined to follow the directions literally, and it is interesting to note that aphasic patients of a higher educational level adopt the latter type of reaction almost as frequently. As a general rule, the degree of scholastic achievement plays the major rôle in the results obtained with these tests, as will be seen from the following: Repetition of the alphabet orally and graphically was done perfectly by all the subjects of group A. The subjects of group B had attained the upper grades of the public school at least, and only one patient made an error. His mistake was the one we had noted frequently in cases of aphasia; he repeated the letters as far as O correctly, but failed badly in the number and sequence of the succeeding ones. He was unable to write the letters further than P. The subjects of group C were of a lower educational level, only one having obtained more than a public school education. Three of six failed to repeat the alphabet, one reaching Q, another R and the third going only as far as C without mistake. Not one of the six wrote the letters correctly. The mistakes occurred in the letters following R, except in the third subject already mentioned, who failed after D. Only one patient in group E wrote and repeated the letters without error. One subject repeated them correctly but failed to write them after T. Another hesitated as to the letters after P, and had to regain the sequence by repeating several short series of letters before he could conclude the recital. The third patient failed in both forms of repetition. He had attended school only until the age of 10. One of the patients in group D had never attended school, so was unfit for the test. The other made no mistakes.

None of the subjects in groups A or B were asked to repeat the alphabet after the examiner, with the exception of the patient who failed to repeat it spontaneously. He made no error. Mistakes were not made by the subjects in group C or by the educated subject in group D. The uneducated subject in the latter group miscalled several letters toward the end of the series. This can be explained best by her retardation, as she was suffering from arteriosclerotic depression. This part of the test was the only one that the general paralytic patient did correctly. Copying the alphabet was done without mistake by the persons in groups A and B. In group C, one of five patients did not make a mistake. The errors made by the other subjects in this class consisted in the omission of one or more letters, but there was no correspondence between different patients as to the letters omitted. This type of mistake is explained best by lack of attention and unfamiliarity with the use of a pencil. Paralysis of the left side makes writing difficult, as there is no way of holding the paper, and these patients had neglected this art for months or years. None of the patients in group E made any mistakes, nor did the subject who was literate in group D. It is evident that this test is one of literacy, but its use gives interesting results in the examination of aphasic persons. One of our patients started the first few letters correctly and then commenced counting, sometimes by ones, sometimes by twos. This form of response has never been noted in our series of control subjects.

Imitation Tests.—The large number of failures made by aphasic patients in the next group of tests was the reason that this study of the responses of nonaphasic persons was begun. These tests—the performance of complicated

acts in response to orders given orally and in writing—are the first group encountered so far that seems difficult, and the difficulty is really greater than it appears at first glance. In this series the patient and examiner face each other. The examiner touches his eye or his ear with either his right or left hand, and the subject imitates his movement. The subject next makes similar movements in response to oral and written commands, and lastly, the examiner makes the motions and the subject tells him what he has done. Head appreciates the difficulty with the first part of the series and supplements it by standing beside his patient facing a large mirror and asking the patient to imitate the movement seen in the mirror. He calls attention to the fact that the phenomenon of reversal, that is, the patient doing exactly the opposite of what the examiner does, is common among normal people. On first impression it seems an easy thing for a person to copy precisely the movement made by some one facing him, but any one who has used tests of this nature will realize the essential difficulties involved. In fact, the recording of this test has been the most difficult part of the whole examination. It is interesting to note that in the Red and Blue song of the University of Pennsylvania, when the cheer leader stood facing the stands and moved to the right, the audience moved to the left, so that orders were given that if the song required a movement to the right, the cheer leader would move to the left and vice versa. However, although Head admits that reversal is a normal phenomenon, he makes the statement that a normal person appreciates when the hand crosses the face to touch a part of the opposite side. It will be anticipated that there was a high percentage of errors made in this group of tests by the control persons.

In the first part of this test—that in which the subject imitates the movement made by the examiner—three of six persons of group A made one error each. This mistake always occurred with the third movement when the examiner touches his left eye with his right hand. This is the first time in the test when the hand crosses the face. This mistake consisted of a complete reversal of the act, that is, the subject touched his right eye with his left hand. One other person in this group made the first two imitations quickly and accurately, but showed great hesitation and slowness in the third act though his result was faultless. Only one of the nine subjects of group B gave a perfect result. Among the rest the number of mistakes of this type varied from one to fourteen out of sixteen imitations.

Of greater interest than the reversal phenomenon is the fact that five of these subjects, in some part of the test, failed to appreciate that the hand had crossed the face to touch the opposite side of the body. For example, if the examiner touched his right ear with his right hand, the subject would touch his left ear with his right hand, or if the examiner touched his left eye with his right hand, the subject would touch his right eye with his right hand. The number of this type of mistake varied from one, which was immediately corrected, to five out of sixteen movements. In group C there was not one perfect score, and in only three was the reversal phenomenon the cause of the failure. The low incidence of this factor in this group is accounted for by the fact that the other four patients had an immovable left arm, and the test had to be modified by using only the unparalyzed side. Every subject in this group made from three to eleven errors in that part of the test which involved decision as to whether the hand did or did not cross the face. One subject made a mistake of a different kind which had been noticed among other aphasic patients. When the examiner placed his right hand on his left eye, the patient touched his right ear with his right hand. Both subjects in group D made many errors. In

the first case every test given was reversed. In the second there were three reversals in sixteen tests, and the patient failed to perceive the crossing of the face five times. The four subjects of group E made errors of the reversal type only. One failed to perceive the hand crossing the face, but immediately corrected this mistake. The general paralytic patient imitated only one movement out of eight correctly. Five times she reversed the examiner's act, and twice she failed to perceive the hand touch the opposite side of the body.

It was felt that some of the subjects were dominated by the strain of being tested and by the fear of failure. It was explained that there was no necessity of hurrying, and that they could have all the time they needed, but even after this explanation the number of mistakes did not diminish. This portion of the test consisted of eight parts, and it was repeated in most of the cases twice. The repetition always showed a distinct improvement as the result of the training during the first part. The large percentage of failures by nonaphasic persons in this test emphasizes the fact that it has no real place in the examination of an aphasic person, for the criteria necessary to judge whether the error of such a patient is due to aphasia or not is lacking. A similar criticism applies to its employment as a test for apraxia.

The results in the second part of this group of tests, namely, the performance of similar acts in response to oral command, are in striking contrast to the first part. This second part has more usefulness as a test of speech disorder, for it requires the ability to understand spoken words and to translate them into motor activity. Our subjects made a few mistakes, however. A real error did not occur among the first group. One person, probably as a result of the strain of the preceding test, touched his right eye with his left hand in response to the order, "Touch your left eye with your left hand," but he corrected his error at once. One subject in group B, when asked to touch his left ear with his left hand, did the opposite. The subjects in group C did not do so well. Three of the six failed once to appreciate the fact that the hand must cross the face, though one corrected his mistake as soon as he made it. One of the three twice did the opposite of what he was asked, though he realized and corrected his error both times. One patient in group D failed twice to touch the opposite side. One patient in group E gave a reversal error that was corrected immediately. The general paralytic patient failed three times to touch the opposite side.

The third portion of this group deals with the ability to read a printed command and follow its directions. This was done by our controls on the whole as well or better than was the preceding section. A curious type of mistake, however, occurred. If the order was to touch the right ear with the right hand, the subject would touch the right eye. The type of test material used may have been responsible for this error. The entire series of orders were printed on one card. It is possible that if each order were on a separate card, the subject would not be so liable to lose the place after executing a command or to confuse the ending of one order with the next. This explanation will not suffice for one person in group B who read the command aloud, "Put your right hand to your right eye," but touched his right ear. This type of mistake occurred in two persons in group A, in five in group B and in four in group C. One of the latter on one occasion also touched the part of the same rather than of the opposite side, and one of the other patients in this group gave a reversal reaction which he corrected. The mistake of touching the eye instead of the ear or vice versa occurred in two patients in group E, though both realized and corrected their mistake. One patient in group D touched the part of the same rather than of the opposite side once, and did the opposite of what had been ordered on another occasion.

The final part of this series combines the difficulties of the first part, that is, the appreciation that the opposite parts of the body are moved with the necessity of translating the movement seen into oral or written speech. We have modified Head's original test by substituting an oral for a written report in order to save time. The subjects of group A found this test as difficult as the first part of the series. Only three of the six did not make mistakes. One subject reported the opposite of what was done. The second failed to realize that the hand had crossed the face, and the third substituted the eye for the ear on two occasions. He had made a similar mistake in executing written orders, and the result in both instances seems to be attributable to carelessness. Four of five subjects in group B made mistakes—the three types of errors—reversals, lack of appreciation of the hand crossing the face and the substitution of the eye for the ear constituting the failures. One patient repeated aloud what he saw, "the left hand touching right eye" but wrote "left hand touching left eye." Group C made a better record than group B, three of six subjects not making mistakes. Only one subject from the last three groups gave a faultless report.

When these tests are applied to patients with aphasia, if the patient is able to understand what is required, precisely the same types of error occur, though possibly in a more pronounced degree. This result would indicate that it is impossible to formulate any theory of the mechanism of speech on a test of this character, and even makes one doubt its applicability to the study of apraxia.

Clock Tests.—This series of clock tests consists of five parts. The subject is required to set the hands of a toy clock in positions similar to those of the hands of a clock placed before him; next, he is asked to tell the time; third, to set his clock to oral command, and lastly, to set the clock to printed commands given first in ordinary nomenclature and then in railroad time. In setting a clock, the hour hand automatically follows the movement of the minute hand, but on these toy clocks it is necessary to set both hands independently. This may be the explanation for some of the inaccuracies observed. There are four typical errors that occur with frequency throughout the series. The first consists in setting the minute hand 5 or 10 minutes before or after the time required, that is, if 10 minutes past 7 were the designated time, the clock would be set at 5 minutes or 15 minutes past 7. Strangely enough, this error also occurred when the patient was asked to tell the time. The second common error was made in setting the hour hand exactly on the hour designated, if the time asked for was between two hours; that is, if 20 minutes to 4 was the time designated, the minute hand would be placed at 8, the hour hand either on 3 or 4, usually the latter. Such a mistake could not be made in the second part of the test. The third common error consisted in reversing the hands. If 5 minutes after 8 was the required time, the minute hand would be placed at 8 and the hour hand at 1, or in the second part of the test the time would be given as 20 minutes to 1. Fourth, the clock would be set one hour before or after the required time, that is, if 25 minutes to 3 was required, the clock would be set either at 25 minutes to 2 or at 25 minutes to 4, or in the second part given as one of these times. The distribution of these types of mistakes is indicated in table 2.

Other varieties of error occurred also. In the first part one patient in group E, shown a clock set at 5 minutes to 2, set his clock at 5 minutes past 2. One patient in group B, shown a clock set at 20 minutes past 11, reported the time as "20, no, 25 minutes to 11." In the second part of the series, a patient in group C reported 8:45 as a quarter to 3, another reported 5 minutes past 8 and 10 minutes past 7 as 5 to 8 and 10 to 7, respectively. One subject in

group D called 20 minutes to 6, 20 minutes past 5. In the third part of the test—setting the clock to oral command—one subject in group A set the clock at 20 to 12 for 20 past 11; one in group C at 20 past 5 for 20 to 6. A similar error was made by one patient in group D. One of the members of group E set both hands of the clock at 8 for 20 to 4. More of these irregular varieties of errors occurred in the final part of the series than in the other portions and involved all of the groups. The commonest form of these mistakes was to place the minute hand on the other side of the clock than it should have been placed, such as setting the clock at 5 to 8 for 5 minutes past 8, or at 20 to 12 for 20 minutes after 11. In some instances the subject read the time or set the clock at 10 minutes past 5 instead of 10 past 7. In others, the time the clock was set was read aloud from the card rather than the time printed thereon. These varieties

TABLE 2.—*The Distribution of the Four Typical Errors in the Clock Tests*

	Imitating Examiner's Clock	Telling Time	Setting Clock to Oral Command	Setting Clock to Printed Command, Ordinary Nomenclature	Setting Clock to Printed Command, Railway Nomenclature
Part 1. Erroneous Setting of Minute Hand					
Group A.	1 error	1 error	0 errors	0 errors	0 errors
Group B.	4 errors	3 errors	0 errors	0 errors	0 errors
Group C.	2 errors	5 errors	1 error	7 errors	3 errors
Group D.	1 error	0 errors	2 errors	0 errors	0 errors
Group E.	5 errors	0 errors	1 error	0 errors	0 errors
Part 2. Misplacement of Hour Hand					
Group A.	1 error	0 errors	0 errors	0 errors	0 errors
Group B.	0 errors	0 errors	0 errors	3 errors	3 errors
Group C.	5 errors	0 errors	2 errors	0 errors	8 errors
Group D.	0 errors	0 errors	0 errors	0 errors	0 errors
Group E.	3 errors	0 errors	1 error	0 errors	8 errors
Part 3. Reversal of Hands					
Group A.	0 errors	0 errors	0 errors	0 errors	0 errors
Group B.	5 errors	3 errors	0 errors	0 errors	0 errors
Group C.	5 errors	2 errors	0 errors	0 errors	0 errors
Group D.	2 errors	0 errors	0 errors	0 errors	0 errors
Group E.	0 errors	0 errors	0 errors	0 errors	0 errors
Part 4. Erroneous Setting of Hour Hand					
Group A.	0 errors	1 error	1 error	0 errors	1 error
Group B.	3 errors	2 errors	1 error	0 errors	6 errors
Group C.	2 errors	0 errors	1 error	3 errors	5 errors
Group D.	0 errors	0 errors	0 errors	0 errors	0 errors
Group E.	2 errors	0 errors	0 errors	1 error	2 errors

of errors were the only ones that we encountered in examining aphasic persons who were cooperative.

Color Tests.—The recognition and naming of colors was done without error by all subjects except one in group C, who, being asked to point to the orange color, pointed to the yellow.

Man, Cat and Dog Tests.—Series 8 and 9 are the first tests that deal directly with words and sentences, and the distribution of errors in our results falls most heavily where it would be anticipated, namely, in the patients with little education and a low intelligence quotient. The first part of series 8 consists in asking the patient to read aloud eight simple phrases, each containing two of the three nouns, dog, cat and man, for example, "the man and the dog," "the cat and the man," etc. One subject in group B read dog for cat. A similar error occurred in one subject in group C. Another in the latter group omitted two of the sentences. These errors are those of attention and might not have occurred had these phrases each been on separate cards. The second part con-

sists in showing the patient pictures of these three objects in order similar to that of the phrases and asking him to read them in a similar manner. No errors occurred in any of our controls. There is one response to this test that occurred rather frequently in the older, less educated subjects. This was to read the pictures from right to left rather than from left to right. As there are three pictures, the same one will recur in each of two successive phrases; these patients named the older picture before the newer one. This form of reaction was made by one subject in group A, two in group B, six in group C, one in group D and two in group E. The third part of the series was to write the eight sentences from dictation. Three types of mistakes occurred, two of them being made by subjects in group C and one in group D. These were reversing the position of the two last phrases, omitting all connecting words and writing gog for dog and reversing the position of the nouns.

The next part is similar to part 2, except that the phrases are written. The same persons who transposed the order of the nouns in part 2 did it here also, though two of the six in group C corrected their former error. One subject in the same group wrote cat for the picture of a dog on one occasion.

The completion of the series consists in copying the phrases from the printed card. This was done poorly by four subjects in group C and by one in group E. One person in group C omitted to write the connecting words "and the." Another lost his place so often that it is difficult to determine from his record what phrases he was writing. A third subject wrote "the cat and the dog" for "the man and the dog." The subject in group E reversed the order of the nouns.

Reading a Paragraph.—Series 9 consists of reading, writing and reproducing orally and graphically a simple paragraph. We used the paragraph giving the story of the fire in New York that is included in the series for 10 years of age in the Stanford revision of the Binet-Simon tests. This was selected because the results are easy to record and the paragraph should not be too difficult for any of our controls. The series consists in having the subject read the selection silently once. In psychometric examinations, the subject reads it aloud and must do so in thirty-five seconds. We did not place a time limit on the reading, as we felt that to do so might disturb the response we desired. The subject is then asked to tell the story in his own words. The Stanford test requires that eight memories be reproduced for successful accomplishment. Only one subject in groups B, C, D and E was able to give more than that number. The largest number of memories reproduced by group A was eighteen. Some of the responses made by the former groups clearly indicated that even the subject matter of the paragraph was not understood.

The second part consists in asking the subject to read the selection aloud. This was done perfectly by all our controls except one patient in group C, who read "the" for "three" and omitted the date and place. The subject is then asked to write the story in his own words. All of our controls were able to reproduce more memories, four in groups B, C, D and E giving more than eight. The highest number in group A was twenty. Finally, the patient writes the story from dictation. Three subjects made mistakes, one in group B omitted many words, one in group C made a similar error, and a second in the same group wrote 5,000 for 50,000.

Complicated Commands.—Series 10 consist of a number of commands, each of which necessitates the performance of two successive actions. In character it resembles some of the tests for aphasia used by Marie, though on the whole the

orders are less complicated than the ones he uses. The instructions are given orally and consist of the following: 1. Give me your hand and put out your tongue. 2. Shut your eyes and put out your tongue. 3. Put out your tongue and give me your hand. 4. Give me your hand and shut your eyes.

These four commands are repeated three times. Simple as the commands appear, only five subjects, 2 in group A and three in group B did them all correctly. As a rule, the mistakes made by the other subjects began with the third order and consisted in doing the three actions—giving the hand, putting out the tongue and shutting the eyes—at once. This type of error was more common in the tests that did not include shutting the eyes, which act was the added feature. The subjects in group A reported that they had the greatest difficulty in controlling this tendency, and even though the overt act was not done, the impulse to do so was felt. It would seem that the associated movement of the muscles that close the eyes and open the mouth are controlled only slightly by the motor area, and a high degree of voluntary inhibition is needed to dissociate them. Whatever the reason for these mistakes, it is plain that this test must not be used in examining aphasic patients, else erroneous conclusions may be drawn from the result.

Drawing Tests.—In series 11 the patient is asked first to draw a common object, usually a bottle, second, an elephant, and third the plan of a familiar room. The results with our series of controls are shown on the accompanying figures.

Coin and Bowl Tests.—The coin and bowl tests used are the following: four glasses are placed before the subject, who is instructed carefully that the order is always from left to right. This is necessary, as we found both aphasic and nonaphasic patients who tended to number them from right to left. A coin is placed in the second glass, and the subject is directed, first orally, then through written commands, to transfer this coin from one glass to another. The first movement is from the second glass to the third, the second from the first to the third. As the coin is already in the third glass, practically every subject hesitated, looked perplexed and finally said it was impossible as the coin was in the third glass. Further instructions had to be given, namely, that the coin could be moved to the first glass and then from the first to the third. This test was well done, only seven subjects, one in group B, five in group C and one in group E making mistakes. Four of these made errors in response to both written and oral commands. The first common mistake was to take the coin from the glass in which it was placed and transfer it to the final glass designated without putting it in the intermediate position. In addition, several of the subjects placed it in the wrong glass; for example, when asked to transfer the coin which was then in glass 1, from glass 3 to glass 1, one subject placed it in glass 4. This type of error occurred with only four patients. The first mistake is not a serious one, and if it is remembered that certain nonaphasic persons do make mistakes and if the rules are made less stringent, this test is well suited to the testing of both aphasia and apraxia.

Geometric Figures.—The final series consists of three parts. The card used for the comparison of geometric form (in the Stanford revision of the Binet-Simon test) was placed before the patient, and a figure similar to one on the card was shown to him. He was asked to point to the corresponding one. Then he was asked to name certain simple geometric figures, such as a square and a circle, and finally, to indicate them on being told their names by the examiner. No errors were made by any subject in this series of tests.

SUMMARY

Our study has pointed out the fact that the normal person often gives responses which are exactly the same as those obtained in cases of aphasia. In some tests, errors are so frequent and coincide so closely with those in aphasic cases that it has been deemed wise to discard these tests. In other tests the mistakes are less frequent but do occur. In these instances a knowledge of the percentage of errors in normal persons makes it possible to evaluate the aphasic response more clearly, and gives an indication of the emphasis to be placed on these tests. It can be said without exaggeration that there is almost no test used in the examination of aphasia in which a normal person will not at some time register errors. Even with people of superior intelligence mistakes are fairly frequent, and they are often exactly the same as those made by patients with aphasia.

The results obtained by examining a series of nonaphasic persons with the tests used by Head for investigating speech disorders show how misleading it may be to draw conclusions from the results of these tests with aphasic persons. In the whole series of thirteen groups of tests, only two, the first and the last, were done without error by our controls. The frequency of errors in series 5, 6 and 10 makes these tests almost worthless as a means of examining speech. Furthermore, the errors noted here are precisely the errors found in the reactions of aphasic patients. It is difficult to explain some of the mistakes in the clock tests, but they seem to be related to handedness and to fall into the category of errors found in series 5. Series 2, 3, 7 and 12 seem the best adapted of the whole battery, with the exception of 1 and 13, for the study of aphasia. It should be noted that the responses of our controls to each of these four groups contain certain errors which discount the absolute value of the test for the study of speech disorders. If an aphasic patient gave the responses of the patient in group C to the second series—that is, on being asked to select a penny he pointed to a pencil, when the penny was placed in his hand out of sight, he selected the pencil as the corresponding object, and when handed a card asking him to select a key, he pointed to the pencil first—it would be taken as evidence of aphasia, and the result would contribute toward whatever theory of speech the examiner was elaborating. Series 8 and 9 are without doubt tests of aphasia, but they are also tests of educational status and intelligence. The alphabet tests come in the same category. The factors involved in the drawing tests are difficult to evaluate, but these tests are more closely allied to tests of intelligence than to tests of the mechanism of speech.

CONCLUSION

The use of Head's tests in our control series has demonstrated to us their value as additional tests in the study of aphasia. Head has unquestionably contributed to the study of speech by the elaboration of his test series. Nevertheless, it is obvious that a more complete outline of examination is necessary than is possible with this series alone, an outline which will deal fully with the manifold aspects of speech.

It is impossible to separate in any way the function of speech from intelligence, as the psychic processes are too subtly interwoven to permit such dissection; but the results with Head's tests on this series of controls indicate that the tests investigate intelligence to a far greater degree than they do speech, and the conclusions drawn from them must stress, automatically, the loss in the former sphere rather than in the latter. In this connection it is significant that we had begun to formulate a theory of speech and disorders of speech on the basis of a series of aphasic persons studied by these tests. An almost identical theory could be developed on the basis of the results with this group of nonaphasic subjects.

While the conclusions reached are based on the tests formulated by Head, we wish to emphasize that any other series of tests would have given identical results. As it is, we consider the tests used by Head as good, if not better, than any others that have been devised for the testing of aphasia.

COLLOIDAL GOLD REACTIONS WITH SPINAL FLUIDS CONTAMINATED WITH BLOOD*

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Lumbar puncture resulting in a bloody spinal fluid occurs not infrequently in general practice and may happen occasionally to the most experienced physician. There are times when a second puncture is inadvisable or refused; yet the Lange test would prove of material aid in making a diagnosis. The following experiments, 214 in number, were undertaken in an attempt to find a way to utilize these contaminated fluids.

In order to produce, experimentally, conditions comparable to those encountered in clinical practice, the patient's blood was added in increasing amount to his own spinal fluid. The maximum amount of blood added was 5 cmm. of blood to 1 cc. of spinal fluid. This produced a fluid definitely pink and corresponding macroscopically to the most contaminated fluid received in the laboratory. A red cell count made on this mixture averaged about 25,000 cells per cubic millimeter of blood.

Normal spinal fluids were used in the first part of the experiment. In series *A*, we added 5 c.mm. of whole blood to 1 cc. of spinal fluid; in series *B*, we added 25,000 washed red cells to 1 cc. of spinal fluid. The number of red cells just equaled those found in 5 c.mm. of whole blood used in the previous experiment. In series *C*, we added the plasma of 5 c.mm. of blood to 1 cc. of spinal fluid. In series *D*, normal spinal fluids alone were used as a control. The results of the colloidal gold reaction with this series is shown in chart 1.

It is evident that both whole blood and washed red cells have practically the same effect on the Lange curve. Both tend to exaggerate greatly the normal curve (*D*) and to produce curves paretic in type, except in the first few tubes. When the red cells were removed, the corresponding amount of plasma still exaggerated the normal curve, but to a much less degree. These results would indicate that if contaminated spinal fluids containing as many as 25,000 red cells per cubic millimeter are centrifugalized, the remaining plasma will not cause a normal spinal fluid to exhibit any change in its Lange reaction other than an exaggeration of the normal curve.

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* Read at the Fifty-Third Annual Meeting of the American Neurological Association, Atlantic City, N. J., May, 1927.

* From the Neuropsychiatric and Clinical Laboratories of the Stanford University School of Medicine.

Abnormal spinal fluids formed the basis of the next experiments. Fluids of the general paralytic type were utilized first; to these were added whole blood, washed red cells and plasma just as in the previous experiments. Colloidal gold tests with these mixtures gave the curves shown in chart 2.

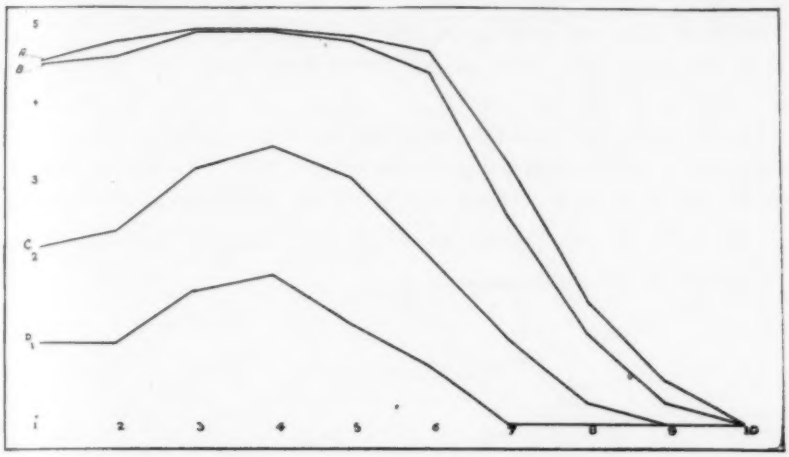

Chart 1.—Curves of negative spinal fluids showing effect of addition of *A*, whole blood; *B*, washed red cells; *C*, plasma; *D*, normal spinal fluids.

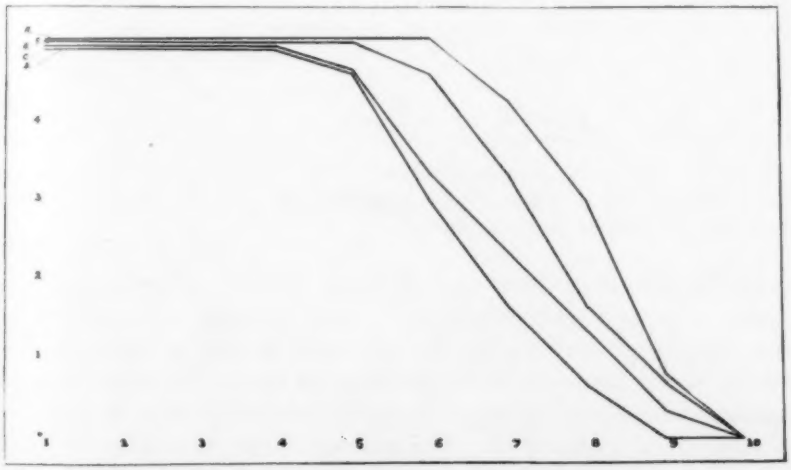

Chart 2.—Curves of general paralytic fluids showing effect of *A*, whole blood; *B*, washed red cells; *C*, plasma; *D*, general paralytic spinal fluids.

These experiments indicate that whole blood and washed cells added to a general paralytic type of fluid result, as might be expected, in a general paralytic type of curve. This curve differed from that produced when normal spinal fluid was contaminated by whole blood only in the

first two tubes. When the red cells were removed, the plasma and general paralytic fluid still gave a general paralytic curve, and one easily distinguished from that of plasma and normal fluid (chart 1, C).

In order to demonstrate the mechanism involved in these reactions, we utilized not only spinal fluids of a frankly general paralytic type, but also fluid of "lower precipitation." The results are shown in chart 3.

In these cases we were again definitely able to show that the plasma merely intensifies the curve of the spinal fluid used. It never changes the character of the curve.

To the syphilitic type of fluids whole blood, washed red cells and plasma were added, as in the previous experiments. Colloidal gold tests gave the curves shown in chart 4. Again the blood and washed red cells

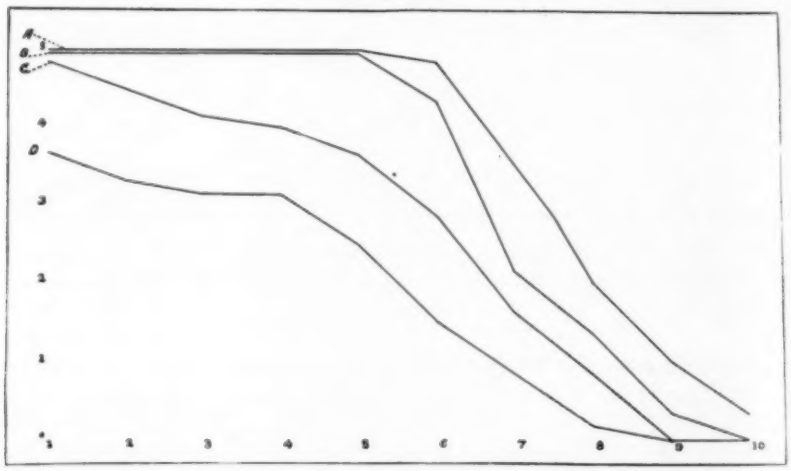

Chart 3.—Curves of incompletely reacting general paralytic fluids showing effect of addition of *A*, whole blood; *B*, washed red cells; *C*, plasma; *D*, incompletely reacting general paralytic fluids.

produced a general paralytic type of Lange reaction. The plasma in this instance, also, only exaggerated the normal syphilitic type of reaction. Therefore, centrifugalizing out the red cells, leaving in the plasma and syphilitic spinal fluid will give a Lange curve easily distinguishable from a contaminated general paralytic fluid, but less definitely separated from a contaminated normal fluid. The distinction can be made only by the height of the curves.

The time element in the spinal fluid contamination was next studied. Normal spinal fluids placed in the icebox, left at room temperature or incubated at 37 degrees C. for four days did not show any change in the colloidal gold curve. Next, whole blood was added to either normal or pathologic spinal fluid, and the mixture was allowed to stand for three days either in the icebox or at room temperature. At the end

of this time, the red cells were removed by centrifuge, and a colloidal gold test was made on the remaining spinal fluid plus plasma. The resulting curves were identical with those shown in charts 1 and 2.

When, however, spinal fluid contaminated with blood was incubated at 37 degrees C. for one day, or even when it was left standing at room

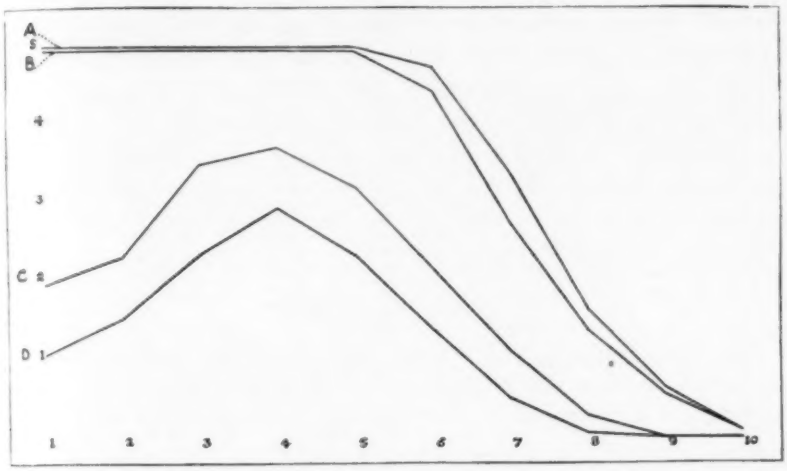

Chart 4.—Curves of syphilitic type fluids showing effect of addition of *A*, whole blood; *B*, washed red cells; *C*, plasma, and *D*, syphilitic spinal fluid.

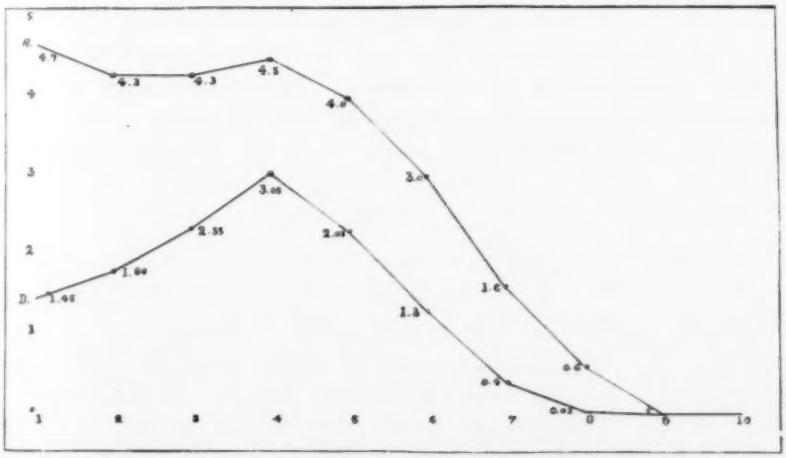

Chart 5.—Curves of fluids syphilitic in type after treatment. *A* indicates plasma curve, and *B*, the curve of fluids syphilitic in type.

temperature for four days or longer, hemolysis generally occurred. The centrifugalized product of such fluids always produced a general paralytic type of curve. The supposition that hemoglobin was the causative agent in producing these curves was proved when hemoglobin derived from

5 c.mm. of whole blood was added to 1 cc. of normal spinal fluid. Fluids so treated gave a general paralytic curve, while the washed stroma of the same number of red cells added to the same spinal fluid did not influence the curve. It appears that spinal fluids, even when contaminated with blood, may be allowed to stand a reasonable length of time without affecting the result of the gold curve, provided hemolysis has not occurred. Blood contamination in spinal fluids rendered syphilitic in type as the result of treatment, produced the type of curve shown in chart 5. On checking over the histories of these cases, it became evident that the original curve was always a general paralytic one. The addition of blood plasma to a spinal fluid of the syphilitic type seemed to cause a reversion to the orignal type in the first two or three dilutions. This phenomenon may be of practical use in differentiating normal fluids from fluids made normal by treatment.

CONCLUSIONS

Spinal fluids which are contaminated with blood, but from which the cells are removed by centrifugalization before hemolysis occurs, produce a colloidal gold curve altered in intensity only; the type of the curve remains the same.

Spinal fluid from patients with general paralysis made normal by treatment, on contamination with blood plasma shows a tendency to revert to the former general paralytic type of curve.

The original colloidal gold curve of spinal fluids experimentally contaminated with the patient's blood can be approximately reconstructed, provided hemolysis has not occurred. There appears to be reason to believe that similar reconstruction can be made of curves of spinal fluids accidentally contaminated.

AN ANALYSIS OF ABNORMAL POSTURE OF THE HEAD IN PARKINSONISM OF VARIOUS ORIGINS *

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Among the most striking and interesting characteristics of patients with parkinsonism of any origin are the postural abnormalities. The most typical disorder is a combination of masklike facies, forward flexion of the head and trunk, flexion at the hips and knees, adduction of the arms and flexion of the elbows and fingers. In addition, flexion of the wrist may be present. This posture was named "dissolution of erectness" by one of us in 1924.[1] This idea was further developed in 1927,[2] and at the same time Brock and Wechsler [3] put forward the thesis that this posture occurred because of disease of the paths underlying the righting reflex which enables erectness to be maintained under normal circumstances.

However, this classic dissolution of erectness does not always occur in all details; the exceptions may be divided as follows: (1) fragments of the complete picture of dissolution of erectness occurring either in the trunk or extremities; these are common. (2) Extension of the trunk rather than flexion, with or without the classic postural defects in arms and legs; these are rare. (3) Various positions of the head, not typical of classic dissolution of erectness, i. e., rotation, lateral flexion, extension or a degree of flexion greater than usual; these are common. It is for the analysis of the last group of cases that this investigation was undertaken.

METHOD OF STUDY

Photographs of patients with parkinsonism, of the degenerative as well as the encephalitic varieties, were taken. Lateral views were found to be reliable for the analysis; front and back views were not reliable.

* From the Neurological Division, Montefiore Hospital.

* Read by title before the Fifty-Third Annual Meeting of the American Neurological Association at Atlantic City, N. J., May, 1927.

1. Kraus, W. M.: Le phénomene de Holt. Les effets pendant la marche du déplacement en avant du centre de gravité du corps sur la position maxima de support de la jambe. La signification à l'état normal et dans les conditions pathologiques, Ann. d. méd. **15**:67 (Jan.) 1924.

2. Kraus, W. M.: Erectness in Man, Arch. Neurol. & Psychiat. **17**:1 (Jan.) 1927.

3. Brock, S., and Wechsler, I. S.: Loss of the Righting Reflex in Man with Special Reference to Paralysis Agitans, Arch. Neurol. & Psychiat. **17**:12 (Jan.) 1927.

owing to difficulty in establishing reliable lines coinciding with the trunk. The rotation of the trunk present makes such lines difficult to determine. Lateral views were carefully traced. Two lines were drawn, one from the external meatus to the outer canthus, another from the external meatus through the trunk and crossing a horizontal line corresponding to the floor. The angle made by the meatus-canthus line with the meatus-floor line will be called the head-trunk angle. The angle made by the

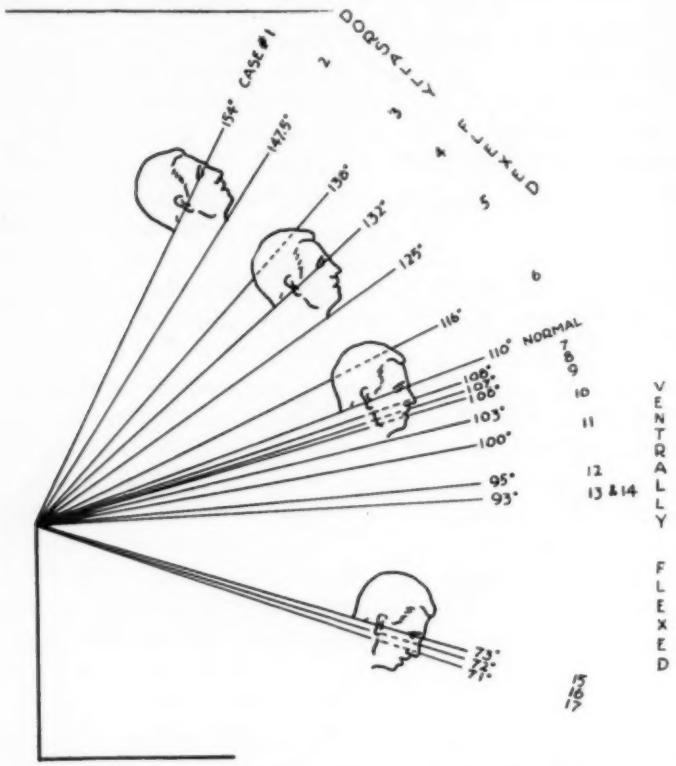

Fig. 1.—Composite view of seventeen patients, showing deviation of the head-trunk angle dorsally and ventrally from the normal position of 110 degrees. Cases 1 to 6 inclusive are examples of the group with epidemic encephalitis.

meatus floorline with the horizontal floorline will be called the trunk-base angle. Each case (figs. 3 to 19) is represented by a tracing on which these angles are given. A brief summary of the case is in the legend. The group of seventeen cases has been represented together for comparison in figures 1 and 2. In the former the head-trunk angles are shown. The normal is 110 degrees. In the latter, the trunk-base angles are shown. The normal is 90 degrees. The accompanying table contains the figures obtained.

Deviations from Normal in the Head-Trunk Angle and the Trunk-Base Angle *

Case	Head-Trunk Angle, Normal, 110 Degrees	Difference from Normal	Trunk-Base Angle, Normal, 90 Degrees	Difference from Normal
1†	154	+44	71	−19
2†	147.5	+36.5	64	−26
3†	138	+28	77	−13
4†	132	+22	78	−12
5†	125	+15	80	−10
6†	116	+ 6	73	−17
7‡	108	− 2	79	−11
8†	107	− 3	80	−10
9†	106.5	− 3.5	76.5	−12.5
10†	103	− 7	76	−14
11†	100	−10	81	− 9
12‡	95	−15	74	−16
13‡	93	−17	43	−47
14†	93	−17	79	−11
15†	73	−37	67	−23
16‡	72	−38	45	−45
17†	71	−39	67	−23

* It should be noted that a head-trunk angle greater than normal occurred only in the encephalitic group (cases 1 to 6 inclusive).
† Parkinsonism—epidemic encephalitis.
‡ Parkinsonism—degenerative.

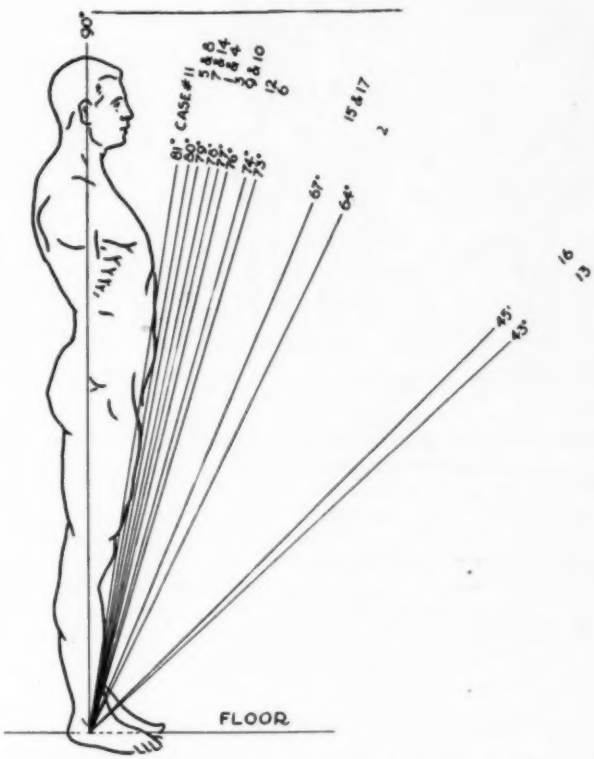

Fig. 2.—Composite view of seventeen patients, showing deviation of the trunk-base angle from the normal of 90 degrees.

COMMENT

By actual measurement, it has been found that the trunk-base angle in our cases was always less than 90 degrees, irrespective of the position of the head. The head-trunk angle, measured laterally, may vary from the normal of 110 degrees in either direction; in our series, it varies in extension to 154 degrees and in flexion to 71 degrees. Of course, each case may show a slightly different angle from time to time under varying conditions. These variations are not great.

In addition to the dorsal and ventral flexion of the head, there is, as a rule, lateral flexion and rotation. Thus the simple flexion forward of the head, such as occurs in pure dissolution of erectness, is obscured

Figure 3

Figure 4

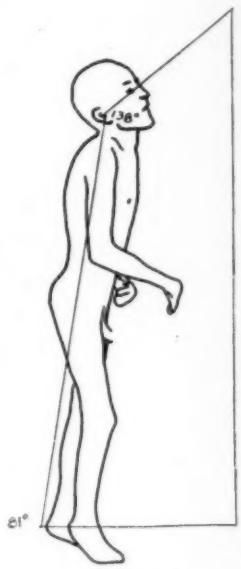

Figure 5

Fig. 3 (case 1).—J. R., aged 36, admitted Feb. 28, 1923. Onset six years before picture was made, with fever, diplopia and lethargy lasting two weeks, soon followed by slowness and stiffness of movement with festination. Condition gradually progressive. Diagnosis: chronic epidemic encephalitis, parkinsonism.

Fig. 4 (case 2).—L. N., aged 35, admitted Sept. 3, 1922. Onset six years before picture was made, with fever, headache, aching pains in extremities and delirium lasting six days, during which time the patient complained of diplopia. Delirium passed into profound lethargy lasting ten days. Two years later tremors, stiffness of gait and slowness of movement became apparent. Diagnosis: chronic epidemic encephalitis, parkinsonism.

Fig. 5 (case 3).—A. H., aged 37, admitted April 6, 1921. Onset six years before picture was made, with fever, aching pains and delirium lasting three weeks. One year later tremors, stiffness of gait and slowness of movements became apparent. Diagnosis: chronic epidemic encephalitis, parkinsonism.

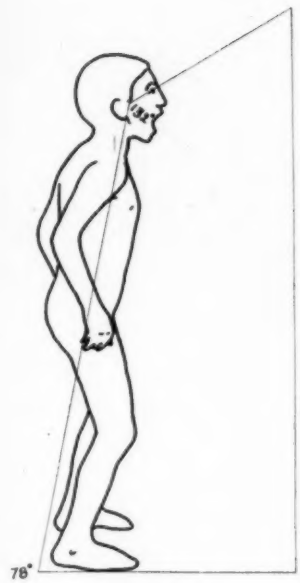

Figure 6

Figure 7

Figure 8

Fig. 6 (case 4).—S. D., aged 11, admitted June 10, 1923. Onset four years before picture was made, with headache, diplopia and abdominal pain. One month later sialorrhea and hyperemotionalism became evident, with attacks simulating decerebrate rigidity. There were athetoid movements of the hands. Since that time there had been a remarkable transformation into an advanced state of parkinsonism. Diagnosis: chronic epidemic encephalitis, parkinsonism.

Fig. 7 (case 5).—T. M., aged 26. Onset four years before picture was made, with diplopia and lethargy lasting one week. Six months later tremors of all extremities and slowness of movement developed. Diagnosis: chronic epidemic encephalitis, parkinsonism.

Fig. 8 (case 6).—H. L., aged 47, admitted June 13, 1924. Onset five and one-half years before picture was made, with aching pains in the extremities. About one year later weakness, stiffness of gait, tremor of hands and indistinctness of speech developed. Condition gradually progressive. Diagnosis: chronic epidemic encephalitis, parkinsonism.

Figure 9

Figure 10

Figure 11

Fig. 9 (case 7).—C. L., aged 76, admitted Aug. 1, 1925. Onset six and one-half years before picture was made, with tremor of the left arm. Very insidiously, tremors and stiffness of the remaining extremities appeared. Patient has advanced arteriosclerosis. Diagnosis: parkinsonism, degenerative.

Fig. 10 (case 8).—H. B., aged 17, admitted Aug. 22, 1926. Onset two and one-half years before picture was made, with restlessness and emotional disturbances. One year later tremors and stiffness of the extremities developed. Patient exhibited a distinct personality defect. After the onset of the disease, patient consumed large amounts of water. Diagnosis: chronic epidemic encephalitis, parkinsonism.

Fig. 11 (case 9).—Y. L., aged 29, admitted Aug. 19, 1922. Onset five and one-half years before picture was made, with lethargy and fever lasting two weeks. Two years later, following childbirth, tremors and stiffness of gait slowly developed. Diagnosis: chronic epidemic encephalitis, parkinsonism.

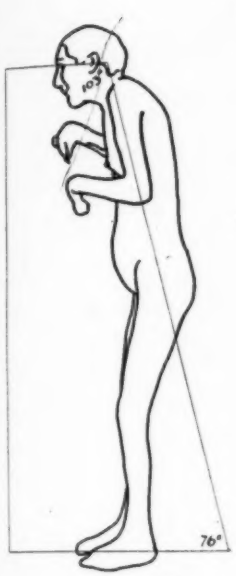

Figure 12

Figure 13

Figure 14

Fig. 12 (case 10).—A. Z., aged 37, admitted May 22, 1922. Onset six years before picture was made, with fever, diplopia and lethargy. A few months later tremor, stiffness of gait and increasing weakness developed. Condition slowly progressive. Diagnosis: chronic epidemic encephalitis, parkinsonism.

Fig. 13 (case 11).—E. H., aged 43, admitted July 20, 1923. Onset six and one-half years before picture was made, with transient diplopia and occasional periods of lethargy. Several months later, tremors, stiffness of gait and slowness of actions gradually developed. Diagnosis: chronic epidemic encephalitis, parkinsonism.

Fig. 14 (case 12).—A. Y., aged 64, admitted May 7, 1925. Onset five years before admission with tremor and stiffness of the left arm. This slowly progressed to involve all extremities. Patient had marked generalized arteriosclerosis. Diagnosis: parkinsonism, degenerative.

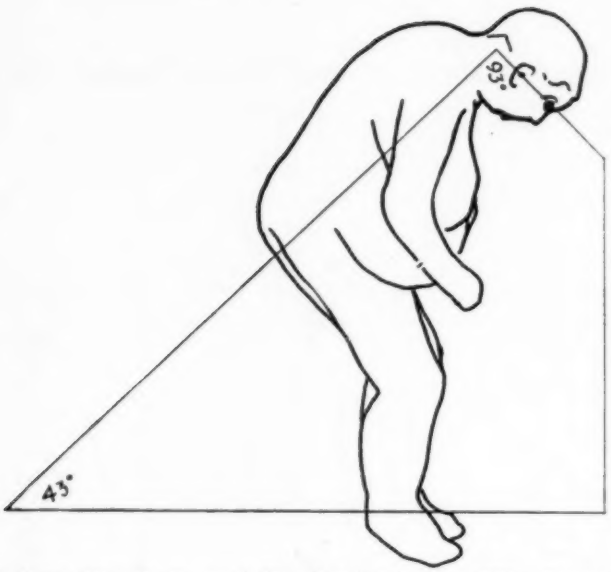

Fig. 15 (case 13).—S. B., aged 57, admitted Aug. 9, 1925. Onset four years before picture was made, with tremor of right hand. Two years later tremor of all other extremities developed with stiffness of gait and festination. She had marked generalized arteriosclerosis. Diagnosis: parkinsonism, degenerative.

Figure 16

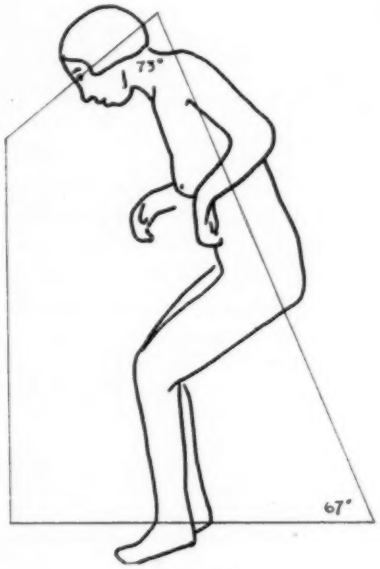

Figure 17

Fig. 16 (case 14).—A. D., aged 24, admitted May 23, 1923. Onset six years before picture was made, with pronounced lethargy lasting three weeks; no diplopia. Two years later slowness and stiffness of movement with tremors developed. Diagnosis: chronic epidemic encephalitis, parkinsonism.

Fig. 17 (case 15).—E. B., aged 27, admitted June 18, 1923. Onset six and one-half years before picture was made, with headache, transient diplopia and lethargy lasting three weeks. Five weeks later, tremor and stiffness developed. Condition slowly progressive. Diagnosis: chronic epidemic encephalitis, parkinsonism.

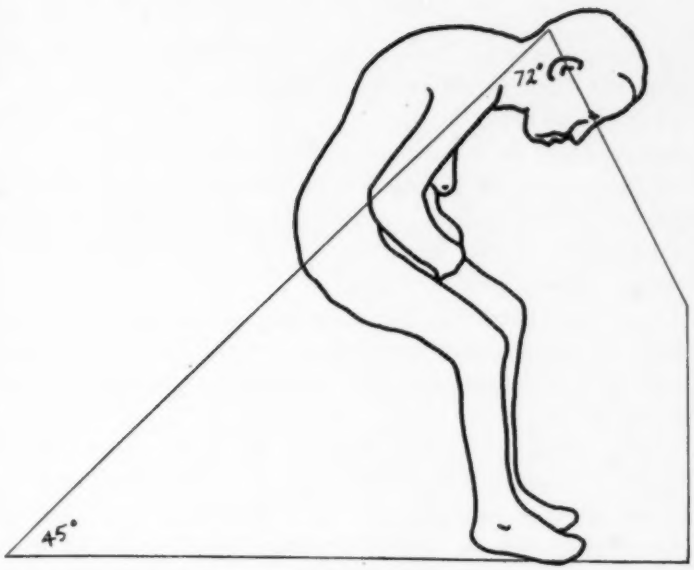

Fig. 18 (case 16).—B. S., aged 74, admitted June 7, 1926. Onset three and one-half years before picture was made, with tremors and stiffness of movement. Condition slowly progressive. There was marked generalized arteriosclerosis. Diagnosis: parkinsonism, degenerative.

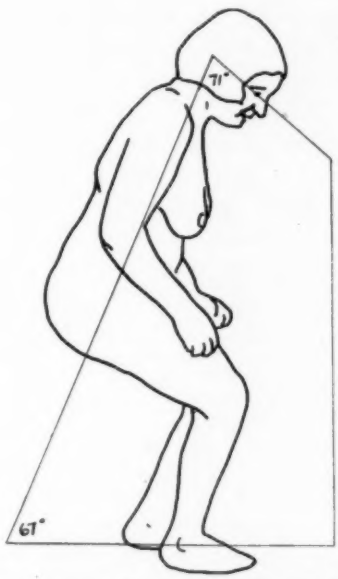

Fig. 19 (case 17).—A. W., aged 19, admitted Jan. 12, 1924. Onset six and one-half years before picture was made, with headache, diplopia and drowsiness. Two months later, she developed choreiform movements. Persistent tremors and gradual stiffness of movement developed. Condition slowly progressive. Diagnosis: chronic epidemic encephalitis, parkinsonism.

by these deviations in all three planes. An increase of the head-trunk angle has not been found in the degenerative types of cases. It is common in the encephalitic type.

We believe that the following conditions may occur:

1. If the paths controlling erectness are symmetrically diseased and the paths controlling posture of the head are not diseased, the classic picture of dissolution of erectness appears. This is found in both degenerative and encephalitic types.

2. If the paths controlling erectness are asymmetrically diseased and the paths controlling posture of the head are not diseased, the head may be rotated or laterally flexed. This is found in both degenerative and encephalitic types.

3. If the paths controlling erectness are either symmetrically or asymmetrically diseased and, in addition, the paths controlling posture of the head are also diseased, the head, in addition to being laterally flexed and rotated, will be overflexed either dorsally or ventrally. This occurs in encephalitis. We have not observed dorsal overflexion in the degenerative variety.

SYMPATHETIC PARALYSIS*

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METHODS OF STUDY

Most clinical studies of anatomic conditions are conducted according to some outlined plan, but the study of the sympathetic nervous system has never been attempted in such a manner. Hitherto, the sympathetic system has been considered in toto and has not been separated into central and peripheral components. The outworn terms vagotonia and sympathicotonia are still being used, and the vegetative nervous system is still being investigated by means of various pharmaceutic preparations.

In the clinic of neurology at the university, we have adopted a method of studying the motility and the reflexes of the sympathetic system similar to that in use for the somatic system. Our investigations have convinced us that the somatic and sympathetic systems have a similar pattern. We began the study with the spinal cord because it alone retains the metameric divisions. We chose cases of paraplegia for study because they show alterations in several metameres, some lying at the level of the injury and others above or below it. A transverse injury of the spinal cord produces not only paraplegia, para-anesthesia and automatism of the reflexes, but also a sympathetic paraplegia. This is manifested by parahypertonia, parahyperviscosity and parahyperglycemia. In paraplegia, it is clear that the blood in the pathologic tissues of the paraplegic area must have physicochemical properties different from those of the blood in the upper part of the body. Our task, then, was to find a method of investigation that would give a clear picture of sympathetic paralysis. The healthy portion of the body was always compared with the unhealthy, and we considered: (1) the properties of the blood vessels; (2) the physical properties of the blood, and (3) the blood chemistry.

PROPERTIES OF THE BLOOD VESSELS

CAPILLAROSCOPY

Method.—The study of the cutaneous capillaries was made by means of a small microscope attached to an adjustable support. Parts of the skin were illuminated by an electric lamp and a lens, while the patient lay on a couch in a dark room.

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Capillary Vessels in Paraplegia.—We succeeded in demonstrating three different types of capillaries in paraplegia: (1) in the healthy skin; (2) below the focus and (3) at the level of the focus. Even if individual discrepancies are admitted, it may be asserted that the capillaries in the healthy skin above the focus are well filled, sinuous and broad; on the background, one can see both arterial and venous loops.

The vessels below the focus are narrow, anemic and spastically contracted. They are seen on a pale ground, and one cannot distinguish a venous from an arterial loop.

Between these two zones is the third, which lies at the level of the focus, occupying one or two segments with indistinct borders. Here sinuosity is replaced by parallelism, and the separate arterial loops vanish. The background becomes somewhat paler than the healthy zone.

Summary.—Below the lesion there is a spasm of the capillary vessels corresponding to the rise of muscular tonus in the somatic sphere. The three zones observed bear a close analogy to the sensory disorders in the somatic sphere, namely, normal sensibility, complete anesthesia and a band of hyperesthesia. Capillary vessels possess their own tonus (Krogh), and this tonus is associated with an unimpaired state of the central vegetative fibers.

Fig. 1.—Capillary conditions in paraplegia. In *A*, showing a normal condition of the capillaries, the darker areas indicate the venules and the small figures, the arterioles. *B* represents the transition zone and *C*, below the focus.

DERMOGRAPHISM

Method.—Dermographic lines of irritation were drawn vertically through healthy and affected parts of the skin along the median, axillary, mamillary, clavicular and vertebral lines of the trunk. The lines were made by means of: (1) the handle of a percussion hammer; (2) a needle (painful irritation); (3) a small, narrow vessel filled with hot water or ice, and (4) a faradic brush. The color of the stripes and their intensity, breadth, swelling, etc., were noted.

Result of Investigations.—In all seventeen cases of paraplegia, certain differences were noted between areas at levels higher and lower than the focus. This difference became perceptible at a certain height, with an interruption between the higher and lower levels. This band of interrupted dermographism was situated two segments higher than the beginning of sensory disorders. At the

same level, a disorder of the perspiration and pilomotor reactions were found. Above this interruption was a bright colored band on the skin—the band of irritation. The dermographic bands, going from above downward, had the following characteristics: uppermost, a pink skin (normal); lower, an intensively colored band, one segment wide (approximately)—a zone of irritation—and often an unaltered zone. Below the latter, the band was somewhat paler than that of the healthy skin. The sensory disorders began two segments lower than the unaltered zone. In cases in which we believed that compression of the substance of the cord existed (in spondylitis) and in which phenomena of irritation (hyperesthesia) were present dermographism below the focus was brighter and redder.

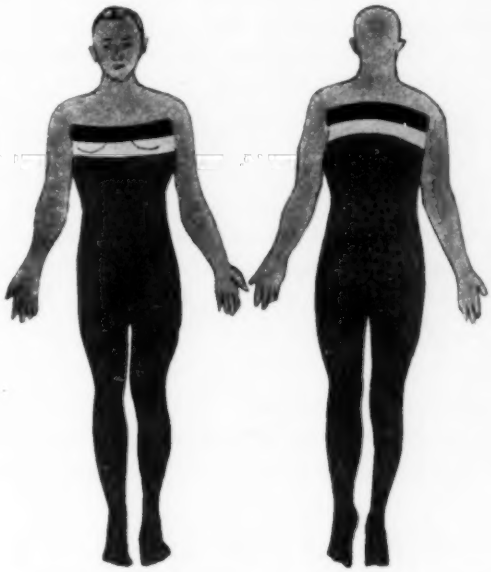

Fig. 2.—Dermographism in paraplegia. Normal dermographism is indicated by the plain area; the area of irritation, by the solid surface (from the second to the third dermatome); the segment of interruption, by the area of cross hatching, and the dotted area indicates that sensory disorders begin at the second dermatone (two segments lower than the zone of interruption).

Summary.—There is a complete analogy to the somatic sphere in compression of the spinal cord. From above downward are present: a band of normal sensibility, a band of hyperesthesia (irritation of the roots) and a band of complete anesthesia (rupture of roots). This analogy may be carried further; the interruption in dermographism corresponds to a definite segment (fig. 2), which lies higher up than the sensory disorders.

Thus, we have a method of objective topical diagnosis based on the level of vegetative disorders.

BLOOD PRESSURE

Method.—The measurement of blood pressure (in the arm and leg) was done with a Riva-Rocci apparatus with Korotkoff's cuff, by auscultation or by palpation. On the leg, the femoral artery was compressed in Hunter's canal, and the tones were auscultated over the popliteal artery. The objective auscultatory method was preferred.

Results.—In a great majority of cases (90 per cent) in healthy persons, the blood pressure in the leg was higher than in the arm; in 10 per cent of the cases, it was equal. In 66.6 per cent of twenty-one patients with paraplegia, the blood pressure in the leg was higher than in the arm; in 19.05 per cent it was equal, and in 14.35 per cent it was lower. The difference in blood pressure between the healthy arm and the diseased leg was 25 and sometimes 50 mm. In patients with paraplegia and isotonia there was a common type of somatic paraplegia. In the third group of patients, who had paraplegia associated with hypotonia, two presented cerebral phenomena (impure cases).

Comment.—Parahypertonia was observed in the majority (66.6 per cent) of cases of paraplegia. The increased tonus arises from spasm of the fibers of the smooth muscles of the vessel walls. We consider this spasm (increased tonus of smooth muscle) analogous to hypertonia in the somatic sphere. It is the result of rupture of the central sympathetic neuron. We consider the fall of blood pressure—parahypertonia—a sign of irritation of the central neuron.

It may happen that in the presence of somatic disorders sympathetic injuries are not present—an isotonia. We believe that all cases showing changed blood pressure, either parahypertonia, parahypotonia or isotonia, will fit in with a somatic conception. As an illustration, one case is reported.

REPORT OF CASE

K., aged 56, with syphilitic myelitis at the level of the first and second dorsal segments, presented sensory disorders on the mesial surfaces of both shoulders. The gait was slightly ataxic; this condition was more pronounced on the right side. The muscles of the right arm were weak. Increased tendon reflexes with a Babinski sign were present on the right. In the circulatory system dilatation of the heart and aorta were present, with a systolic blood pressure of 185 in the right arm; 187 in the right leg; 165 in the left arm, and with 205 in the left leg.

There was a difference between the two sides of the body. The left half (in which somatic disorders were fewer) would correspond exactly with the 66.6 per cent of our cases. On the right half, isotonia was present (the pressure was absolutely increased). The difference between the arms is explained by the presence of an aneurysm of the aorta. The striking difference between the left arm and leg is explained by the localization of the focus on the left side.

PHYSICAL PROPERTIES OF THE BLOOD

GENERAL PROPERTIES

Technic of Withdrawal of the Blood.—The ideal method of obtaining samples of blood is by venipuncture, but it is difficult to secure blood from a vein in the leg in paraplegia. The veins protrude less distinctly in such cases and the blood clots more rapidly; the conditions of withdrawal of the blood from veins of the arm and leg are therefore different. Before the finger, arm or leg is punctured, it is necessary to massage the area lightly. Frank's needle is used.

Appearance of the Blood.—The blood from the arm appears rapidly (in one or two seconds) and flows for a long time; it runs slowly from the leg, and flows for a shorter time. The blood from the arm is bright red; that from the leg is darker.

CLOTTING OF THE BLOOD

Method and Normal Standards.—Determination of the rate of clotting was made by means of the Sitkovsky coagulometer. In ten healthy persons the rate of clotting was the same in blood from the leg as in that from the arm (from three to four minutes).

Clotting in Paraplegia.—Of twenty-five cases of paraplegia the rate of clotting was greater in blood from the leg in 76 per cent. Sometimes this rule obtained only on one side. In 16 per cent of the cases, the rate of clotting of blood from the leg was less. The last group (8 per cent) included two cases in which there was acceleration of clotting in the blood from the leg on one side and retardation on the other.

Summary.—In the majority of cases (76 per cent) of paraplegia, there was acceleration of blood clotting from the leg, and in 100 per cent there was unequal coagulation time between the blood from the arm and that from the leg.

VISCOSITY

Method and Viscosity in Healthy Persons.—In ten healthy persons, the viscosity of the blood in the arms and legs coincided. In these investigations, viscosity was measured by Hesse's viscosimeter.

Viscosity in Paraplegia.—In nineteen cases of paraplegia it was found that: (1) in 52.6 per cent the viscosity of blood from the leg was less than that from the arm (parahypoviscosity); (2) in 21.1 per cent, the viscosity was equal (isoviscosity); (3) in 21.1 per cent, the viscosity from the leg was greater than from the arm (parahyperviscosity), and (4) in 5.8 per cent hypoviscosity was present on one side of the body and hyperviscosity (vegetative asymmetria) on the other. It is of interest to note that vegetative asymmetria corresponds to the somatic Brown-Séquard syndrome.

Comparison of Viscosity and Clotting.—Normally, variations in viscosity and clotting run parallel. We did not observe such parallelism in our cases. A similar absence of parallelism has been pointed out in hemophilia, and it is possible that our observations on sympathetic phenomena may throw some light on the nature of this disease.

DRIED SOLIDS

Method.—Equal quantities of blood (5 cc.) from the veins—mediana cubiti and dorsalis pedis—were poured into two small weighed glasses, each containing some distilled water, and the glasses reweighed. The glasses were then put into an oven for five hours at from 60 to 70 C.; they were then placed in an incubator at 37 C. until perfectly dry, and then reweighed. The percentage of dried solids was calculated for the whole blood taken for the experiment. The conditions of withdrawal, drying and weighing of the blood were the same for the arm as for the leg.

Results.—In ten cases of paraplegia the weight of the dried solids was greater from the leg than from the arm, in 70 per cent; in 20 per cent, it was equal, and in 10 per cent it was less from the leg (spondylitis with hyperesthesia).

Comment.—The change in weight of the dried solids and other changes of the blood are results of metabolic disturbances in the tissues. Disturbance of normal circulation (vascular spasm) causes some arrest of metabolic products in the tissues, and this is reflected in the weight of dried solids. A certain density of blood in connection with diffusion of metabolic products is probably regulated by nervous impulses in the walls of the vessel. There are no central impulses, the wall of the vessel permitting only the passage of water; the denser parts of the blood are arrested in the plasma. We think that these factors play an important part in the variations of concentration of the blood in paraplegia.

CHEMISTRY OF THE BLOOD IN PARAPLEGIA

BLOOD SUGAR

The influence of the vegetative nervous system on the metabolism of carbohydrate may now be considered established. The muscles and skin contain about 70 per cent of the glycogen in the organism. It is possible, then, that in a transverse lesion of the spinal cord the structures below the focus of injury are deprived of the regulating influence of the brain, and the content of sugar in the blood, above and below the focus, a priori, should be different.

Method.—Estimation of sugar in the capillary blood above and below the focus was carried out simultaneously by Hagedorn-Jensen's method.

Results.—In healthy persons, variations in blood sugar in the arm and in the leg were as high as 10 per cent; this was considered, therefore, to be within the limits of experimental error. In paraplegia the difference in sugar content of the blood from the arm and that from the leg reached 23 per cent. From this standpoint the twelve cases of paraplegia may be divided into four groups: (1) parahyperglycemia, embracing 50 per cent of all cases (there is more sugar in the blood from the leg); (2) parahypoglycemia, 16 per cent; (3) hyperhypoglycemia, 8 per cent (asymmetria), and (4) isoglycemia, 25 per cent.

Summary.—In one case of parahyperglycemia, an almost complete transsection of the spinal cord was revealed during operation; conse-

quently, parahyperglycemia is a symptom of interruption. To the same group also belongs a case of the somatic Brown-Séquard syndrome in which parahyperglycemia was found on the side of the sensory alterations.

Thus, paraplegia may be accompanied by para-anisoglycemia. We explain isoglycemia by the absence of injury to fibers having a relation to carbohydrate metabolism. Irritation seems to produce increased absorption of sugar by the tissues—a parahypoglycemia. Para-anisoglycemia may also fit into our somatic conception.

Besides the life of a tissue, we must also consider the vascular influences. The wall of the blood vessel separates two colloidal systems—the one liquid (blood) and the other solid (tissue). In a functional disorder of this wall, there must be an alteration in the metabolism of carbohydrates. The quantity of blood sugar, therefore, is determined by two factors: the chemistry of the tissues and the vascular influences.

SUMMARY AND CONCLUSIONS

Attention is called to the rapid change of the properties of the blood when it passes from one level to another. This phenomenon becomes comprehensible when it is realized that the blood, before mixing, reflects

Sympathetic Manifestation in a Healthy Man and in a Case of Paraplegia

	Healthy Soldier, Aged 22		Patient B., Aged 24; Paraplegia; Myelitis Syphilitica	
	Above the Focus—the Arm	Below the Focus—the Leg	Above the Focus	Below the Focus
Capillaries	Broad	Broad	Broad	Narrow
Dermographism	Red	Red	Red	Pale
Blood pressure	116	120	105	145
Blood clotting	3 min. 10 sec.	3 min. 10 sec.	3 min. 50 sec.	3 min. 20 sec.
Viscosity of blood	1/6	1/6.2	1/5	1/4.5
Dry solids, per cent	24.0	24.3	2.4	2.7
Blood sugar	130	122	129	156

the properties of the vessel and the tissue through which it runs. As an illustration, we report two cases in the accompanying table. In the first case, that of a healthy person, the blood pressure, the type of capillaries, the viscosity, the clotting and the blood sugar were equal in the arm and in the leg. This is an instance of complete isotonia.

In the second case, a transverse injury of the spinal cord, definite changes were noted. In the somatic sphere, this patient had a paraplegia; in the sympathetic sphere he had a parahypertonia, a parahyperglycemia, etc. The distribution of the symptoms is correspondingly similar.

THE TWENTY-SIX NORMALLY POSSIBLE FORMS OF ROTATIONALLY INDUCED NYSTAGMUS*

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In a prevous paper,[1] I made an analysis of ampullar stimulation and individual eye muscle response in rotary and in vertical nystagmus induced by rotation, which may be seen in figure 1. The usually

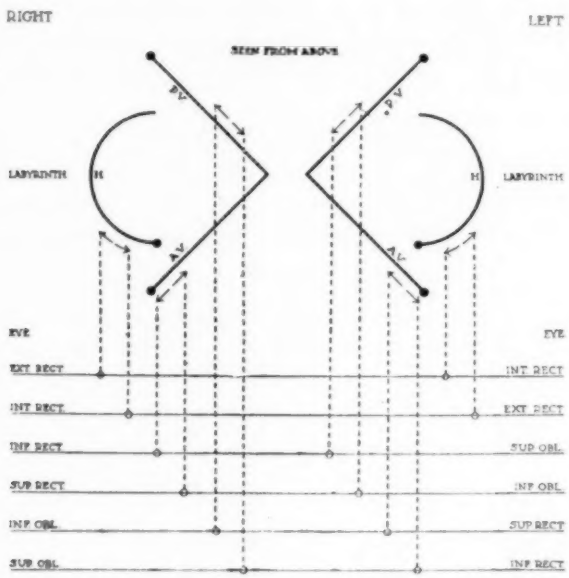

Fig. 1.—The relation of individual semicircular canal currents to eye muscle pairs.

accepted data for horizontal nystagmus are included. With the use of these data, figure 2 was prepared to show the details of the fourteen recognized types of nystagmus that can be induced by rotation.

If it is admitted that rotation produces any movement in the endolymph, either current, slight shift or merely migration of heavier molecules or ions, then the positions and rotations that are used should affect the canals as indicated by the arrows. The muscles thus stimulated,

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* Read at the Seventh Annual Meeting of the Association for Research in Nervous and Mental Disease, New York, Dec. 28, 1926.

1. Favill, John: An Explanation of the Mechanism of Induced Rotary and Vertical Nystagmus, Arch. Neurol. & Psychiat. **13**:479 (April) 1925.

according to my previous analysis, are the muscles needed to produce the clinical phenomena that are known to occur.

As both labyrinths are fixed in the same skull, a canal-current picture in one labyrinth determines the canal-current picture in the other labyrinth. The positions used in producing all the recognized types

RECOGNIZED TYPES OF NYSTAGMUS INDUCED BY ROTATION

POSITION OF HEAD	ROTATION	CANAL CURRENTS AFTER ROTATION		EYE MUSCLES STIMULATED		RESULTING SLOW COMPONENT	SYMBOL FOR SLOW	SYMBOL FOR QUICK
		RIGHT	LEFT	RIGHT	LEFT			
30° FORWARD	R	POSTERIOR / SEEN FROM ABOVE / ANTERIOR		EXT.RECT.	INT.RECT.	HORIZONTAL TO R.	←	→
30° FORWARD	L			INT.RECT.	EXT.RECT.	HORIZONTAL TO L	→	←
60° BACK OR 120° FORWARD	R			SUP.OBL. SUP.RECT.	INF.OBL. INF.RECT.	ROTARY OVER TO L	↷	↶
60° BACK OR 120° FORWARD	L			INF.OBL. INF.RECT.	SUP.OBL. SUP.RECT.	ROTARY OVER TO R.	↶	↷
90° BENT TO R. SHOULDER OR 90° BENT TO L. SHOULDER	R			INF. OBL. SUP.RECT.	INF. OBL. SUP.RECT.	VERTICAL UP	↑	↓
90° BENT TO R. SHOULDER OR 90° BENT TO L. SHOULDER	L			SUP.OBL. INF.RECT.	SUP.OBL. INF.RECT.	VERTICAL DOWN	↓	↑
30° FORWARD AND 45° BENT TO R. SHOULDER	R			INF.OBL. SUP.RECT. EXT.RECT.	INF.OBL. SUP.RECT. INT.RECT.	DIAGONAL UP & R.	↖	↘
30° FORWARD AND 45° BENT TO R. SHOULDER	L			SUP.OBL. INF.RECT. INT.RECT.	SUP.OBL. INF.RECT. EXT.RECT.	DIAGONAL DOWN & L.	↘	↖
30° FORWARD AND 45° BENT TO L. SHOULDER	R			SUP.OBL. INF.RECT. EXT.RECT.	SUP.OBL. INF.RECT. INT.RECT.	DIAGONAL DOWN & R.	↙	↗
30° FORWARD AND 45° BENT TO L. SHOULDER	L			INF.OBL. SUP.RECT. INT.RECT.	INF.OBL. SUP.RECT. EXT.RECT.	DIAGONAL UP & L.	↗	↙
75° FORWARD	R			INF.OBL. INF.RECT. EXT.RECT.	SUP.OBL. SUP.RECT. INT.RECT.	HORIZ. R. & ROTARY R.	⇄	⇄
75° FORWARD	L			SUP.OBL. SUP.RECT. INT.RECT.	INF.OBL. INF.RECT. EXT.RECT.	HORIZ. L. & ROTARY L.	⇄	⇄
15° BACK	R			SUP.OBL. SUP.RECT. EXT.RECT.	INF. OBL. INF. RECT. INT.RECT.	HORIZ. R & ROTARY L.	⇄	⇄
15° BACK	L			INF.OBL. INF.RECT. INT.RECT	SUP.OBL. SUP.RECT. EXT.RECT.	HORIZ. L. & ROTARY R.	⇄	⇄

Fig. 2.—Recognized types of nystagmus induced by rotation.

except horizontal nystagmus are such that both vertical canals should be stimulated in each labyrinth.

If one bears these two statements in mind, the canal-current pictures of, say, the right labyrinth alone may be considered. It will be seen on careful examination that only fourteen combinations of currents are possible.

The last two types in figure 2, horizontal plus reverse rotary, are usually not mentioned in textbooks on otology. I predicted them in advance of producing them, but a description of them was subsequently found in Politzer.[2]

A further check on the foregoing assignment of eye muscles to ampullar stimulation in man was desired. It seemed that the steps called for were to: (1) obtain a patient with one dead labyrinth; (2) obtain his consent to the administration of ether for experimental purposes; (3) rotate him, when anesthetized, in positions calculated to stimulate only one vertical canal; (4) note the resulting deviations that should occur, since the quick components are abolished by anesthesia, and decide which eye muscle was responsible in each eye after each rotation. As the second step of this program was not feasible, some other method of checking was sought.

A study of the diagram of the canals shows that there are only twenty-six current pictures possible in one labyrinth. Fourteen of

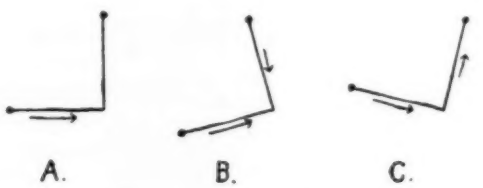

Fig. 3.—Difficulty of producing current in only one vertical canal. Canals are shown edgewise, and rotation has been made in the horizontal plane in the same direction for each case: *A*, with one vertical canal plane exactly in the horizontal plane; *B*, with one vertical canal plane on each side of the horizontal plane (rotary nystagmus type); *C*, with both vertical canal planes on same side of the horizontal plane (vertical nystagmus type).

these have already been accounted for in the recognized types of nystagmus. Can the twelve remaining current pictures with their resulting forms of nystagmus be produced by rotation in the proper positions? Each would call for a current in only one vertical canal of each labyrinth, with or without currents in the horizontal canals.

If one experiments with a glass model of the labyrinth containing some fluid with particles in suspension to show movement, it appears difficult to produce a current in one vertical canal alone. Running upward and inward from their ampullae, the vertical canals of one labyrinth approach each other in perpendicular planes. At their mesial ends, they unite in a common crus, which then continues to the utriculus. With one vertical canal placed in the horizontal plane, the

2. Politzer, A.: Diseases of the Ear, ed. 6, revised by M. J. Ballin, Philadelphia, Lea & Febiger, 1926, p. 168.

other vertical canal will be perpendicular. Rotation in the horizontal plane should not produce a current in the second canal. However, with any slight deviation of the first from the horizontal plane, some stimulus for current production will be present in the second. This is shown in figure 3.

THEORETICALLY POSSIBLE TYPES OF NYSTAGMUS INDUCED BY ROTATION

POSITION OF HEAD	ROTATION	CANAL CURRENTS AFTER ROTATION (RIGHT / LEFT)	EYE MUSCLES STIMULATED (RIGHT)	EYE MUSCLES STIMULATED (LEFT)	RESULTING SLOW COMPONENT WHEN LOOKING TO (RIGHT / CENTER / LEFT)	RESULTING QUICK COMPONENT WHEN LOOKING TO (RIGHT / CENTER / LEFT)
60° BACK THEN INCLINE HEAD AS IT IS—45° RIGHT	R	POSTERIOR / SEEN FROM ABOVE / ANTERIOR	SUP. RECT.	INF. OBL.	↑ / ↷	↓ / ↶
60° BACK THEN, ETC. 45° RIGHT	L		INF. RECT.	SUP. OBL.	↓ / ↶	↑ / ↷
60° BACK THEN, ETC. 45° LEFT	R		SUP. OBL.	INF. RECT.	↷ / ↓	↶ / ↑
60° BACK THEN, ETC. 45° LEFT	L		INF. OBL.	SUP. RECT.	↶ / ↑	↷ / ↓
75° FORWARD THEN, ETC. 45° RIGHT	R		INF. OBL. EXT. RECT.	SUP. RECT. INT. RECT.	↔ / ↘	↷ / ↘
75° FORWARD THEN, ETC. 45° RIGHT	L		SUP. OBL. INT. RECT.	INF. RECT. EXT. RECT.	↻ / ↓	↺ / ↑
75° FORWARD THEN, ETC. 45° LEFT	R		INF. RECT. EXT. RECT.	SUP. OBL. INT. RECT.	↓ / ↺	↑ / ↻
75° FORWARD THEN, ETC. 45° LEFT	L		SUP. RECT. INT. RECT.	INF. OBL. EXT. RECT.	↗ / ↻	↙ / ↶
15° BACK THEN, ETC. 45° RIGHT	R		SUP. RECT. EXT. RECT.	INF. OBL. INT. RECT.	↕ / ↻	↓ / ↺
15° BACK THEN, ETC. 45° RIGHT	L		INF. RECT. INT. RECT.	SUP. OBL. EXT. RECT.	↘ / ↻	↖ / ↷
15° BACK THEN, ETC. 45° LEFT	R		SUP. OBL. EXT. RECT.	INF. RECT. INT. RECT.	↔ / ↙	↶ / ↗
15° BACK THEN, ETC. 45° LEFT	L		INF. OBL. INT. RECT.	SUP. RECT. EXT. RECT.	↻ / ↕	↺ / ↓

"RIGHT" OR "LEFT" ALWAYS MEANS THE PATIENT'S.
ARROWS FOR COMPONENTS REPRESENT ACTUAL MOVEMENTS OF PATIENT'S EYES (←—=TO RIGHT)
←+ OR +→ MEANS HORIZONTAL SLOW COMPONENT IS ABOLISHED BECAUSE EYES ARE VOLUNTARILY TURNED IN ITS DIRECTION.

Fig. 4.—Theoretically possible types of nystagmus induced by rotation.

When one works with a patient, this difficulty may be overcome by approaching the ideal position from two opposite extremes and gradually narrowing them down until the proper position is obtained, much as one focuses a microscope.

A second real difficulty lies in the fact that one never knows exactly in what planes the canals in a given patient lie. This may be partially met by testing the patient to find at just which head positions pure

horizontal and pure rotary nystagmus may be obtained. Positions for new current combinations may then be calculated with more hope of definite results.

The necessary calculations [3] of position and rotation for the twelve remaining canal-current pictures were made in advance, as shown in figure 4.

The eye muscles which, according to figure 1, should thus be stimulated were next filled in.

On attempting to produce these effects with careful placement and retention of the head, it was found that there would result, with the eyes in the center position, only a brief nystagmus of two or three jerks after rotation ceased. Furthermore, this brief movement was small, indefinite and difficult of observation or description.

It was then thought that perhaps with voluntary turning of the eyes to the extreme right or left, those expected slow components which

TABLE 1.—*Functions of Individual Eye Muscles in Three Positions*

Muscle	Eyes Right	Eyes Forward	Eyes Left
Right sup. rectus	Elevation	Up, in and rotation left	Rotation left
Right inf. rectus	Depression	Down, in and rotation right	Rotation right
Right sup. obliquus	Rotation left	Down, out and rotation left	Depression
Right inf. obliquus	Rotation right	Up, out and rotation right	Elevation
Left. sup. rectus	Rotation right	Up, in and rotation right	Elevation
Left inf. rectus	Rotation left	Down, in and rotation left	Depression
Left sup. obliquus	Depression	Down, out and rotation right	Rotation right
Left inf. obliquus	Elevation	Up, out and rotation left	Rotation left

arise from the superior and inferior recti and oblique muscles might be more easily observed.

3. These directions for head placement are in a simplified form. For example, the fifth type in figure 4 calls for the head bent 75 degrees forward, then inclined 45 degrees to the right and the patient rotated to the right. This is an abbreviation of the following steps: (1) 120 degrees forward. (Both horizontal canals are now perpendicular. Each vertical canal is 45 degrees to the horizontal plane.) (2) 45 degrees to the patient's right. (Both horizontal canals are still perpendicular. The right posterior vertical canal and the left anterior vertical canal are now in the horizontal plane. The right anterior vertical canal and the left posterior vertical canal are now perpendicular.) (3) 45 degrees back. (Both horizontal canals are now 45 degrees to the horizontal plane. The right posterior vertical canal and the left anterior vertical canal and are now 45 degrees to the horizontal plane. The right anterior vertical canal and the left posterior vertical canal are still perpendicular.) One hundred and twenty minus 45 equals 75, which is the total degrees of forward bending required.

The same canal-current picture may also be obtained by: 60 degrees back, 45 degrees left and 45 degrees back, with the patient rotated to the left. This means that the head must be back an uncomfortable angle of 105 degrees and is hence omitted. Similarly, in each of the remaining types in figure 4; an alternative but uncomfortable position has been omitted.

Table 1, compiled from data in Fuch's textbook,[4] shows that simplified actions of these muscles predominate when the eyes are turned to the right or left. Using this table, I entered in figure 4 the symbols for the slow components to be expected in the twelve instances with the eyes to the right and to the left, making twenty-four predictions in all.

Using a patient in whom I had produced normally all the fourteen recognized types of nystagmus and whose canals were apparently in the average positions,[5] I tested him in each of the twelve new positions, first with eyes voluntarily turned to the right after rotation and then with eyes similarly turned to the left.

In all twenty-four tests, the nystagmus was exactly as predicted.

To say that one cannot see how endolymph can move is not equivalent to seeing that it does not move. These experiments strengthen the theory that there is some endolymph movement in definite relation to the plane of rotation. Until some better explanation is forthcoming, which will be equally consistent under twenty-six circumstances, simple canal currents afford the best explanation for the ampullar stimuli which cause nystagmus after rotation.

It is believed that these observations corroborate my previous assignment of eye muscle pairs to individual canal currents so far as is experimentally justifiable in man.

122 South Michigan Avenue.

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4. Fuchs, E.: Text-Book of Ophthalmology, trans. by A. Duane, ed. 8, rev., Philadelphia, J. B. Lippincott Company, 1924, chapter 17.

5. Horizontals inclined down and backward 30 degrees. Verticals perpendicular to horizontals and each at 45 degrees to the sagittal plane.

Clinical and Occasional Notes

BARBITAL (VERONAL) ADDICTION

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An increase in the rate of admissions to hospitals for "barbital poisoning" and an observation of the diagnostic pitfalls and social problems of this condition justify inquiry into the situation. This study is based on the reports of 100 patients, with acute and chronic forms of the poisoning, who were seen consecutively at the Denver General Hospital or in private practice during the last three years.

Barbital [$CO(NHCO)_2C(C_2H_5)_2$], diethyl barbituric acid, is a ureid derived from diethylmalonic acid and urea, which was introduced as a hypnotic in 1903. Under the trade name of veronal it now has a tremendous and increasing lay popularity for self administration. Its habit-forming propensity (barbitalismus or veronalismus) became known shortly after its introduction. This fact alone merits the attention of physicians and of the law. In the United States it is usually sold without restriction, promiscuously, as a "safe" hypnotic. It is efficient, its sales "repeat," and it has a sufficient toxicity to cause Great Britain to require record of its sales in the "poison book."

That barbital is definitely habit forming has become increasingly evident for years. Officers who enforce narcotic laws, practicing physicians, individual users and the rate of admissions to hospitals all testify to its steady increase in popularity. It is used alone or in association with whisky or morphine. It is taken as a hypnotic or as a "bracer" following symptoms resulting from debauch or deprivation. It has a certain vogue for suicidal purposes. Long continued use not infrequently engenders a mental or moral obliquity of forensic interest.

In common with most persons addicted to drugs, a majority of barbital addicts are probably found in the lower strata of the community, but not all users are of the "submerged tenth." No level of society is exempt. The factor determining habitual use is more in the individual personality than in the surroundings. Sands[1] classified the persons who were acutely poisoned as of the manic-depressive group and the chronic users as among the psychopathically inadequate or inferior group. My own observation leads to the conclusion that, aside from a few bona fide accidental overdosages, these unfortunate persons practically without exception have some psychic stigma. Many are, or have been, addicted to the use of alcohol, and the majority use tobacco.

Finding himself unable to react adequately to environmental conditions, the man who is either constitutionally or acutely below the standard dulls his sensorium with barbital, just as other drugs are used as a refuge from circumstance. It should be remembered that a host of proprietary hypnotics now on the market are of the same chemical series as barbital and may induce in greater or less degree the same results. Poisoning resulting

1. Sands, I. J.: Barbital (Veronal) Intoxication, J. A. M. A. **81**:1519 (Nov. 3) 1923.

from barbital taken in conjunction with one of its congeners is more difficult of treatment and offers a poorer prognosis. In my experience, phenobarbital is the usual associate.

Chemically, barbital belongs to the methane series, in the alcohol-chloroform group. In explanation of its hypnotic action, Meyer and Overton[2] offered the hypothesis that this series has greater affinity for cholesterin and lecithin than for water and that it tends to accumulate in nerve cells rich in these elements. As these lipoids are more or less dissolved, their function is interfered with and narcosis results. In the case of motor cells, disordered reactions likewise result. If one assumes this hypothesis, it is understandable that excessive dosage or continued administration might induce degenerative changes, possibly permanently, as ethyl alcohol does in like circumstance.

The drug is eliminated through the kidneys as barbituric acid, which is recoverable from the urine. This test is of value in disputed diagnoses. Fischer and Hoppe have recovered barbituric acid from the urine forty minutes after oral administration, but the maximum elimination is not attained till the second or even the third day.[3] In minimal dosage, as much as 90 per cent may be recovered in the urine, but in excessive administration an uncertain but larger amount is retained in the tissues.

The average minimal lethal dose is said to be 50 grains (3.25 Gm.),[4] but tolerance varies greatly. Death has been reported from as little a dose as 15 grains (0.972 Gm.) and many times recovery has followed the ingestion of more than 125 grains (8 Gm.). One man whose case will be reported later, took 100 grains (6.5 Gm.) a day for six days; another, an average of 50 grains for three weeks or more. Both lived, but six months later they still showed mental deterioration.

Pathologic changes seen at postmortem are definite but "not characteristic."[5] Generalized hyperemia is found, more especially of the meninges, liver and kidneys. Edema of the lungs is common; terminal bronchopneumonia is not an unusual symptom. Cardiac dilatation is frequently seen, and fatty degeneration of the liver is not rare, occurring in chronic cases. Renal degeneration is found especially in the tubules, though it is not limited to them. Death is caused by central respiratory paralysis and may be sudden. Barbituric acid may be recovered from all the tissues.

This report deals especially with states of chronic intoxication, but a brief summary of the symptoms found in severe acute poisoning is not amiss. Without a definite history of the patient having taken the drug, diagnosis is uncertain. An actual pathognomonic sign is not present. The sensorium may show any degree of clouding. Respiration is feeble and shallow, and the pulse is slow, thin and irregular. Blood pressure is lowered. The pupils are disordered as to size and reaction, with a tendency to hippus; nystagmus is often present and ophthalmoplegia not uncommon. The temperature is usually depressed and rarely is slightly elevated. The skin is apt to be cold, clammy and mildly cyanotic. Sphincteric control is occasionally lost, but this is probably due more to stupor than to true paralysis. The tendon reflexes are irregularly disordered. Facial asymmetries, hemipareses and ptoses are not uncommon and

2. Meyer and Overton: Studien u. d. Narkose, Jena, 1901; Ergebn. d. Physiol., 1901, p. 666, vol. 2.

3. Fischer and Hoppe: München. med. Wchnschr. **56**:1492, 1909.

4. Willcox: Lancet **2**:734, 1913.

5. Schneider: Prag. med. Wchnschr. **32**:17, 1907. Huseman: Viertel Jarsch. ger. Med. **50**:43, 1915.

later disappear. Confusion, disorientation, thick scanning speech and defects of attention, retention and memory are present. Judgment is poor and insight worse. Especially unfavorable signs in prognosis are respiratory embarrassment, shallow or Cheyne-Stokes breathing, feeble intermittent radial pulse, absent knee reflexes and unduly prolonged deep coma. Patients who are in coma more than five days rarely survive. Treatment at this stage is symptomatic.

The chronic effects of barbital fall into two groups: (*a*) symptoms persisting after acute intoxication, which usually soon disappear; (*b*) those following habitual or long continued indulgence.

The outstanding symptoms of chronic intoxication are both physical and mental. The diagnosis, however, may not be clear because of the patient's wilful misstatements for the purpose of concealment of the infirmity or because of true amnesia due to the toxic state. Hospitalization and serology are often required to differentiate general paralysis, alcoholic pseudoparesis, multiple sclerosis or tumor of the brain, which poisoning caused by veronal may often slavishly imitate. There is much to be said in palliation of mistaken diagnoses on initial examination.

In chronic intoxications stupor is not so frequent or so profound as in acute cases. Its place is taken by psychic aberrations. Generalized cyanosis of low degree, frequently with erythematous eruptions of the skin, is common, though not universal. The gait may suggest cerebellar ataxia but does not show the constancy of the direction of fall. The station is uncertain. General dyssynergia and a coarse waggling incoordination are present. Apraxia is common. Speech is monotonous and scanning, with elisions and transliterations. Handwriting is of the toxic tremor type and shows elisions and reduplications. The pulse is slow and soft; blood pressure is low, and the patient is dyspneic on slight exertion and tires easily. Constipation is usually present. Incontinence is a frequent symptom, but apparently is due to hebetude rather than to paralysis. Urinalysis shows a small quantity of albumin and casts; frequently, hematoporphyrin is found. Reflex disturbances, usually of the hypotonic type, are customary.

Mentally, the picture is one of confusion and uncertainty. Ideation is rambling and incoherent. Thought processes are retarded and incomplete. Defects of attention, retention and association are constant and marked. The ethical sense is dulled, leading to social errors or even to obscenity and indecent exposure. Vague unorganized and unsystematized delusions are common, usually with a paranoid trend. However, one occasionally finds a moderate euphoria. Judgment, orientation as to time, and insight are probably the worst damaged of the psychic faculties and are the last to clear up in convalescence. Cases of residual defect fall almost entirely in this class.

The mentality in this condition precludes complicated criminality or conspiracy, and the appearance does not inspire confidence sufficient for the consummation of any but the crudest frauds or clumsy impositions on friends. Ethical offenses, as against decency, and minor frauds, such as issuing fundless checks and forgery of an unskilful type, are well within the possibilities. Carelessness may well result in acts of an apparently criminal complexion. The paranoid reactions are usually too fleeting to endanger any one, but the dulled sensorium may lead to quasicriminal complaisance with the schemes of others.

Satisfactory treatment outside an institution is difficult or impossible. Danger to life is small except by complications such as suicide, while the

patient is depressed, pneumonia and exhaustion, or following injury resulting from lack of coordination. The patient must be put to bed, with special attention to elimination and supportive treatment. Absolute rest is essential; a bitter tonic given as a routine greatly helps the impaired digestive tract. Fluid intake, with due regard to renal irritation, must be encouraged. Tub baths at 98 F. for from one to three or more hours seem ideal for insomnia and restlessness. If the patient has adequate care, recovery may be expected in from one to two months, though in a few cases certain intellectual defects persist indefinitely; occasionally, great improvement is not noted, though this is rare. The drug may be withdrawn at once or gradually, depending on the condition of the patient. My own preference has been for immediate withdrawal and supportive tonic treatment afterward.

Four cases from this series are cited, each illustrative of special points of history or symptomatology.

REPORT OF CASES

CASE 1.—*Death following acute overdose; autopsy.*

A. R., a white laborer, aged 45, was admitted to the hospital at 4 p. m. and died at 6 p. m. of sudden respiratory collapse. He had been found unconscious at 3 p. m. after one and one-half days' absence from his work. Two empty bottles labeled "veronal" were beside him. They had once contained 100 grains each.

Nothing was learned of the patient's past history. On admission, he was in a state of collapse and was cyanotic and cold, with Cheyne-Stokes respiration. The pulse rate was 60 and intermittent. Systolic blood pressure was 90; diastolic, 55. All tendon reflexes were absent; he was incontinent. Four hundred and fifty cubic centimeters of thin greenish fluid was taken from the stomach by tube; the stomach was washed out, and symptomatic treatment was instituted. At 6 o'clock, he suddenly went into respiratory failure and died. Analysis of the contents of the stomach showed much barbituric acid. The tissues were not tested for it. Autopsy disclosed a "wet brain," pulmonary edema, acute dilatation of the heart, especially of the right ventricle, and some congestion of the kidneys and of the lower end of the stomach and of the duodenum.

CASE 2.—*Uncomplicated barbitalismus in an exalcoholic patient, with hallucinations and delusions. Initial diagnosis, general paralysis. Partial recovery.*

F. C., a married barber, aged 48, began to take barbital three months before admission to the hospital, because of pain caused by an injury to the back. In his younger days he had been addicted to alcohol and had taken a "cure" in 1914; he had not had alcohol since. He had gradually increased the dosage of barbital because it "had lost its effect" and he was unable to sleep. Memory was almost a total loss; he was irritable and so tremulous that he could not work. He lost weight, and the speech became "thick." He developed vague unsystematized ideas of his wife's infidelity. When first seen, he was taking 50 grains of barbital a day and had done so for at least three weeks. He had recently been arrested for alcoholism three times. Examination showed coarse incoordination of the hands, fibrillary tremors of the face, exaggerated and unequal knee reflexes, marked Romberg sign and ataxia. Speech was of the classic "paretic" type. The pupils were unequal, irregular and fixed to light. There was no history of syphilitic infection, and the patient denied having taken barbital recently, though he said that he had taken it "as needed" for years for headache. The tentative diagnosis was general paralysis, and he was

sent to the hospital where the blood and spinal fluid were found normal. Under rest and elimination, the condition cleared up in many respects, but until the day he left the hospital, the man continued to allege that certain events were "framed ups" and that things he claimed to have seen out of the window were obviously hallucinations.

CASE 3.—*Chronic addiction in a person of psychopathic inferior type, with permanency.*

C. L., a widower, aged 48, who for years had been a "promoter" of shady stocks, was said always to have been moderately hypomanic, overenthusiastic, garrulous and tricky, if not actually dishonest. Following the death of his wife a year previously, he was unable to sleep and took barbital. He had continued it practically steadily up to the time he was seen. Two weeks prior to examination he had increased the amount suddenly and markedly, because he was worried over the outcome of a trial for fraud for which he had been indicted. He had been under medical care for about a week, but several times had eluded his attendants, gone to town, filled in counter checks and persuaded friends to cash them. With the proceeds, he bought barbital. He did not have funds in any bank. He wanted to smoke all the time, was careless, and twice had set the bed on fire; he was not disturbed by this and in fact denied that it had ever happened. He was incontinent. He talked in a rambling way of a new oil scheme that he was going to promote when he "got out of this." He resented restraint and cursed the physicians for their interference. The physical signs were like those observed in case 2, but this man complained bitterly of insomnia and wept copiously when he saw the nurse, who he thought resembled his dead wife (she did not, in any respect). He improved slightly for a week, eluded the nurse, went out on the street in his bed gown, was arrested for indecent exposure and was sent to a state hospital, where he remains a patient with classic, moderate organic dementia. Inquiry disclosed that for the four days prior to the first examination he had taken at least 100 grains each day and probably sometimes more. He had taken barbital daily for a year.

CASE 4.—*Psychic shock, the taking of barbital, alcohol, phenobarbital, moral depravity, business incapacity. Recovery.*

Mrs. J. D., aged 29, had seen her husband killed in a gruesome manner three months previously and could not sleep thereafter without a nightmare. Her physician prescribed phenobarbital, to be self administered. As she had always been addicted to the use of alcohol, she took more and more. She discontinued taking phenobarbital alone, so far as I could learn, and took a mixture of barbital and phenobarbital in unknown amount; her apartment was littered with empty containers of both. She became associated with a man interested in closing up her husband's estate and made sexual advances, which he accepted; she became shameless in her demonstrations of affection until relatives intervened. Irritability, total loss of insight and resentment of interference were the outstanding mental features, but physically she was so weak that she could not walk without falling; several times she sustained severe injuries by getting out of bed. After long institutional care she recovered, apparently completely, without insight into her past state. She is reported to be doing well.

Barbital is a danger to the community without restriction of its sale. Addiction to barbital should be considered in cases with a doubtful diagnosis. The prognosis should be guarded as to restoration of psychic faculties, and guardianship should be considered in doubtful cases.

324 Metropolitan Building.

CIRCUMSCRIBED SUPPURATIONS OF THE SPINAL CORD AND MENINGES

Report of a Case of Subdural Abscess with Functional Recovery Following Operation *

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The literature on circumscribed suppurative conditions of the spinal cord is meager. Either local suppuration of the spinal cord and meninges is exceedingly rare, or else the condition is seldom seen at a time when a diagnosis is possible. From our review of the literature, however, it seems likely that circumscribed purulent conditions of the spinal cord and the meninges probably are more frequent than supposed. There are undoubtedly some cases of lowly virulent infection which are not recognized during the acute stage. Some cases progress to a chronic myelitis or to formation of cysts of the leptomeninges and at times syndromes of tumor of the cord occur. The more acute fulminating cases lead to death. A few of these have been recognized at necropsy.

REPORT OF CASE

Clinical History.—F. S., a man, aged 32, whose family and past history were unimportant, was first seen Oct. 7, 1926. He had enjoyed normal health until the fall of 1920, when he developed a cervical furunculosis. At this time, he received a slight trauma to the region of the left hip. This was followed almost immediately by severe pain in the distribution of the left sciatic nerve. The patient was treated for a month for sciatic neuritis in spite of a high temperature. He continued to have pain and lameness of the left leg, but returned to work. In September, 1921, a tumefaction appeared in the left iliac region, which was supposed to be a lipoma; this was removed, but a discharging sinus remained. Osteomyelitis followed. Numerous operative treatments were carried out each year until February, 1926. Since that time, the patient has been free from osteomyelitic symptoms.

About Sept. 1, 1926, he developed pain in the upper dorsal region, which radiated anteriorly to the midthoracic region on the left side. This pain was severe at times and was increased by straining and coughing. On September 27, the patient first noted difficulty in walking; this was followed by paresthesias in both legs. These symptoms were progressive, and involvement of the sphincters developed. The weakness of the legs developed into complete paraplegia. He was admitted to the University Hospital on October 7.

Physical and Neurologic Status.—The patient presented a toxic appearance from chronic cachexia, but the general visceral examination gave essentially negative results. A slight febrile reaction and moderate leukocytosis were present. The neurologic examination gave normal results above the trunk. From the nipple line down, on both sides, hypalgesia and hypesthesia were present, with the same diminution in sensation in the lumbar distribution. The sacral segments were unaffected. Motor weakness was marked in both legs. The abdominal reflexes were absent; the knee and ankle reflexes were

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* Reported before the Douglas County Medical Society, April 12, 1927.

* From the Neurologic and Neurosurgical Departments of the University of Nebraska College of Medicine.

exaggerated. Babinski's toe sign was absent. Vibration and sense of position were lost on both sides. An area of tenderness to percussion was present over the fourth dorsal spinous process.

A combined lumbar and cisternal puncture revealed a complete spinal block. The lumbar pressure was 16 mm. of mercury and was not increased by jugular compression, while the cisternal pressure was 6 mm. and was elevated to 10 mm. by jugular compression. The lumbar fluid was yellowish, with a total protein content of 450 mg. per hundred cubic centimeters. One and five-tenths cubic centimeters of iodized oil 40 per cent was injected into the cistern. Under the fluoroscope, the oil was seen to settle down to the level of the fourth dorsal spine, where it divided on either side of the midline, but none went to a lower level. Thus the upper level of the block was sharply demarcated, as seen in figure 1.

From the history of the chronic recurrent suppurative infection of the pelvic bones and the fever and leukocytosis, an infectious process of the spinal cord seemed probable. The definite segmental level of the symptoms, the root

Figure 1 Figure 2

Fig. 1.—The upper level of the block at the fourth dorsal vertebra.

Fig. 2.—Appearance of the iodized oil 40 per cent after it has settled in the caudal region following operation.

pain, the presence of a spinal block, and the localization shown by iodized oil led to a diagnosis of abscess affecting the spinal cord at the fourth and fifth dorsal vertebrae. It was thought that the abscess was extradural and that the cord symptoms resulted from pressure. In this we were in error.

Operative Report.—The spines and laminae of the third, fourth and fifth dorsal vertebrae were removed. There was considerable bleeding from the epidural space and from the cut laminae, although no definite inflammatory tissue could be identified. The dura was tense and firm for about 3 cm. in the area exposed. A midline incision was made in the dura, and translucent granulation tissue was encountered, not easily differentiated from soft tumor tissue. This was gently explored with a blunt dissection in an attempt to identify the cord and nerve roots. During dissection in the right upper angle, a few drops of creamy pus were released. No cerebrospinal fluid had thus far escaped, and for fear of spreading infection to the meninges, no further dissection was done. The dura was left open and the wound closed with

silkworm gut retaining sutures and interrupted chromic catgut, a rubber tissue drain being bent over the cord and out of the lower angle of the wound. Cultures from the pus obtained grew *Staphylococcus aureus.*

Progress.—After severe postoperative shock from hemorrhage and chronic sepsis the patient made an excellent recovery. Rectal and bladder control returned first, and gradually the motor power was regained, and the sensory symptoms improved. About six weeks after admission, the patient walked out of the hospital using a cane.

An examination on March 14, 1927, showed evidence of a sensory segmental level by partial loss of tactile, pain and temperature sensation; this loss of sensation was much less than before the operation. The patient was still ataxic in both legs, but the sense of position had improved. Vibration sensibility was still absent. The reflexes were normal, with the exception of the plantars and abdominals, which were absent. The patient walked as far as 2 miles a day. Physiotherapy and exercises were carried out, with improvement in the ataxic condition. Diathermy treatments were also given over the site of the inflammatory process. The chronic osteomyelitis was quiescent. Examination on May 14, 1927, did not show any disturbance in epicritic sensibility; the evidence of sensory level had entirely disappeared. The residual condition was a mild chronic meningomyelitis involving principally the posterior columns. Figure 2 shows the iodized oil settled in the caudal sac. The roentgenogram was taken shortly before the patient's discharge from the hospital.

Woltman and Adson[1] recently reviewed all cases of apparently true abscess of the spinal cord. They found only twenty-nine cases, of which all except one (besides their case) had a fatal termination. Their patient made an excellent functional recovery following evacuation of the abscess. Most of the patients in the cases reported have had an associated suppurative spinal meningitis. In a few cases, the condition has been circumscribed, true abscess of the spinal cord. The diagnosis has rarely been made preoperatively or ante mortem. Woltman and Adson stated that Nothnagel alone has made the diagnosis.

Since Woltman and Adson's article was published, Sittig[2] has reported a case of metastatic suppurative pachymeningitis interna with an abscess of the spinal cord following a septic abortion. The spinal fluid was normal, but intracisternal injection of iodized oil revealed a spinal occlusion at the fifth dorsal level. A preoperative diagnosis of tumor was made, but at the first operation diffuse extradural suppuration was found. At a second operation, subdural extension of the pus was present. At necropsy, the pus was found also in the spinal cord. Sittig's case was the first one in which iodized oil had been used as a diagnostic aid in a suppurative condition of the spinal cord. Our case is the second instance in which this form of myelography has been used to localize a suppurative process.

Elsberg[3] stated that abscess of the spinal cord may be secondary to a spinal trauma, or that it may be metastatic from a suppurative lesion in some other

1. Woltman, H. W., and Adson, A. W.: Abscess of the Spinal Cord. Report of a Case with Functional Recovery After Operation, Brain **49**:193 (June) 1926.

2. Sittig, O.: Metastatic Spinal Cord Abscess from Septic Abortion, Ztschr. f. d. ges. Neurol. u. Psychiat. **107**:146, 1927.

3. Elsberg, C. A.: Diseases of the Spinal Cord and Its Membranes, Philadelphia, W. B. Saunders Co., 1916, p. 289.

part of the body. Abscess of the spinal cord soon gives rise to purulent meningitis and usually presents symptoms of acute transverse myelitis. He states that if a diagnosis of an abscess of the spinal cord could be made, the treatment would be surgical, suggesting that a diagnosis is practically impossible.

There have been five reported instances of operations for suppuration of the spinal cord — those of Cavazzani,[4] Spiller,[5] Woltman and Adson[1] and Sittig[2] and our own. Two of the patients have survived, with good functional recovery; one recovered but was totally paralyzed below the level of the lesion, and two cases terminated fatally.

Oppenheim,[6] in the 1911 edition of his book, barely mentioned circumscribed spinal meningitis. In the last edition,[7] he devoted a chapter to chronic adhesive spinal meiningitis and gave a bibliography of the reported cases. He dealt with the residual formations of arachnoidal cysts and adhesions from previous localized spinal meningitis. These cystic and adhesive conditions frequently produce syndromes of tumor of the cord from compression. The increased space given in the discussion of this condition illustrates that lately it has been more accurately diagnosed.

Since the use of the valuable technic of the combined cisternal and lumbar puncture of Ayer and, in selected cases, the use of iodized oil, we have additional means for differentiating these compression or block syndromes from the myelitic group. In reported cases of acute myelitis, we find that the data are incomplete, in many instances particularly with reference to studies of the spinal fluid. It is possible that some of these conditions have been local suppurative lesions.

In the literature on spinal meningitis[8] are reports of a few cases of extradural suppurations. Spiller[5] reported an interesting case of extradural abscess of the middorsal region following a cervical boil. Definite symptoms of spinal block were present, and a preoperative diagnosis was made by aspiration of pus through a lumbar puncture needle at the site of the block. Laminectomy was performed, but the patient died from purulent meningitis. Spiller stated that this was the third case of the kind he had seen. The clinical picture of our patient was similar to that in his case, and our preoperative diagnosis was extradural abscess.

Hinz[9] reported a postpartum case of purulent parameningitis. A definite sensory and motor level was present. The spinal fluid was normal; no block

4. Cavazzani, G.: Abscesso centrale traumatico del midollo spinale operato e guarito, Riv veneta di sc. med. **30**:481, 1899; quoted by Woltman and Adson (footnote 1).

5. Spiller, W. G., and Wright, W. M.: Extradural Abscess of the Midthoracic Region of the Spinal Canal Secondary to a Boil in the Neck, Arch. Neurol. & Psychiat. **5**:107 (Jan.) 1921.

6. Oppenheim, H.: Textbook of Nervous Diseases, translated by A. Bruce, Edinburgh, Otto Schulze & Co., 1911, pp. 298, 310.

7. Oppenheim, H.: Textbook of Nervous Diseases, Berlin, S. Karger, 1923, pp. 419, 440, 443-447.

8. Since this paper has gone to press, we have discovered a recent review of the literature by Dandy (Abscesses and Inflammatory Tumors in the Spinal Epidural Space. [So-Called Pachymeningitis Externa], Arch. Surg. **13**:477 [Oct.] 1926). He collected reports of twenty-five cases of acute epidural infections. Two patients recovered following drainage.

8. Hinz: Case of Purulent Parameningitis, Deutsche med. Wchnschr. **47**:1229 (Oct. 3) 1921.

was present. The antemortem diagnosis was postpuerperal myelitis. At necropsy, the extradural space was filled with thick pus, and intradural infiltration was not present. Hinz stated that Oppenheim reported five cases of extradural suppuration, Morawitz three cases, and Shlick one case.

Braun[9] stated that the epidural space can be infected from: (1) disease of the vertebral column with accumulation of inflammatory products in the epidural space; (2) inflammation, which may extend along the sheaths of the spinal nerve through the spinal foramina to the epidural space; (3) metastatic infectious suppurations which take place within the epidural space, and which do not have any connection with the spinal cord. The case of Hinz is an example of the last. Braun reported two cases: one was a case of metastatic infection from a wound in the shoulder in which signs of intense spinal meningitis developed; the studies of the spinal fluid were negative. At necropsy, the entire epidural space was filled with pus. The second case was one of lower virulence, a metastatic infection from an abscess near the left sacro-iliac joint. The infection traveled by way of the lumbosacral nerves and produced signs that Braun interpreted as an epidural meningitis. In addition, the left peroneal muscles were paralyzed. The results of studies of the spinal fluid were also negative in this case. The patient made a complete recovery.

We did not find any cases classified as subdural abscess in the literature. We believe that these cases have been classified with abscesses of the spinal cord.

COMMENT

We are inclined to believe that while suppurative conditions of the spinal cord are rare, they are not as infrequent as is supposed. We believe that the therapeutic outlook is not necessarily hopeless, and that a diagnosis can be made if the infectious history is kept in mind and careful studies of the spinal fluid are made. The history of a suppurative process elsewhere in the body, the development of a rapidly progressive horizontal spinal lesion, presenting symptoms of spinal occlusion such as increased protein content or xanthochromia of the spinal fluid, a positive Queckenstedt's sign and in certain cases myelography with iodized oil, should suggest the possibility of a suppurative condition of the spinal cord. The association of a syndrome of spinal compression with fever and leukocytosis makes a tentative diagnosis of circumscribed suppuration of the spinal cord probable and indicates the necessity of an immediate laminectomy.

Suppurative diseases of the spinal cord may be classified as follows: (1) extradural (suppurative pachymeningitis externa): (*a*) diffuse purulent epimeningitis — normal spinal fluid; (*b*) circumscribed abscess — producing compression symptoms with evidence of spinal block; (2) subdural (suppurative pachymeningitis interna): (*a*) diffuse spinal leptomeningitis and (*b*) circumscribed abscess, both producing symptoms of block, and residual meningomyelitis, cystic or adhesive; (3) intramedullary: (*a*) multiple abscesses and (*b*) circumscribed abscess, both producing syndromes of block or acute transverse myelitis.

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9. Braun, H.: Spinal Epimeningitis, Zentralbl. f. Chir. **49**:1274 (Sept. 2) 1922.

Abstracts from Current Literature

CRITICAL REVIEW OF THE METHODS OF EXAMINATION OF THE VESTIBULAR APPARATUS. ADDRESS AT THE SECOND ITALIAN CONGRESS ON OTO-NEURO-OPHTHALMOLOGY, ROME, OCT. 20-22, 1926. BARRÉ, Riv. oto-neuro-oftal. **3**:496, 1926.

Barré devotes nearly fifty pages to an exposition of present day knowledge and methods used in neuro-otology. He cautions against forming a judgment based on one examination alone and urges that the caloric, electric and rotatory tests be employed routinely. He finds, however, that the methods of application of these various tests are not at all uniform, and that in many instances authors have reported results without defining the method, therefore leading to confusion. He proposes in this conference to lay down a plan which will meet with the approval of those present and lead to international adoption — a plan for the systematic examination of the vestibular apparatus. A general consideration of these tests shows the following points.

The test of choice should be the least disturbing to the patient. It should bring into play the smallest number of elements, and apparently it should give preference to static over dynamic tests. Moreover, the apparatus employed should be as simple as possible because "as one knows, abundance is not always synonomous with wealth." Before any instrumental examinations are made, the patient is tested in various ways. The presence of vertigo may be obtained from the history, and it seems useful to retain the term "subjective" for the sensation of rotation in the head and "objective" for that in which the objects seem to swim.

In the case of vertigo, it seems to the author that this symptom is entirely labyrinthine, and that it is present in cases of cerebellar disease only when the labyrinthine portions of the cerebellum are disturbed. The first test to perform in cases of suspected labyrinthine disturbance is the Romberg test. Some of the patients are notoriously unstable and they often fall in a fashion similar to that of the tabetic patient. There are, however, differences of importance. The tendency to fall comes on after a notable period and is more apt to be associated with inclination to the left or the right side. It seems feasible, therefore, to differentiate the tabetic from the vestibular Romberg sign. In certain instances the patient may stand well with the eyes shut, and then if the test is made more difficult, such as by bending forward or backward, he tends to fall. In Barré's opinion, however, such increased sensibility, rendering the test positive too often, diminishes its value coincidently.

In his practice the author directs the patient to place his feet parallel and 2 or 3 cm. apart. By means of a plumb line hanging from a cross-arm directly in front of the patient, small deviations can more readily be observed. After the simple standing reaction has been completed, further tests are made with the arms. In the first instance, these are extended directly in front at the level of the shoulder. Sometimes it is useful to have the patient's shoulders supported in order to determine how much of the movement is due to the trunk and how much to the arms. Weakness on one side (from pyramidal disease) brings about lowering of the arm, but, unless there is an added vestibular factor, this lowering is vertical. In cases of vestibular disease, the arm deviates to one side or the other. The duration of the test should be much longer than that ordinarily used, preferably from forty to sixty seconds, but beyond this point fatigue becomes manifest. In doubtful cases it may be repeated several times. Various methods of graphic representation of this deviation have been devised. During the test it is important to keep the head absolutely straight and the eyes closed but looking straight ahead. The position of the head has a good deal of influence on the deviation of the arms.

The pointing tests of Bárány are also useful, pertaining more to the dynamic than the static character. These should also be standardized if possible, and graphic representation is often useful. The significance of past-pointing Barré believes to be labyrinthine. During these tests the normal attitude of the head should be noted, for there are often characteristic displacements. Finally, in the lower limbs there are also pointing reactions which are not well understood. In cases of positive postural deviation there seems to be an active contraction of the muscles on the side toward which the body is bent which is resisted by the other muscles. It is not to be looked on as a more instability of equilibrium.

Finally, the reactions of the eye muscles are examined before any instrumental tests are done. The presence of conjugate deviation, of impaired lateral or vertical movements, or of defective convergence should be noted. Diplopia is a common symptom in acute vestibular conditions, and it is possible to produce diplopia by galvanic stimulation of the vestibular apparatus.

In regard to nystagmus, the author takes the same position as he does toward the Romberg test: marked lateral rotation of the eyes brings out too many jerky movements in normal persons. In normal subjects these movements are unequal, few and of short duration. They reappear irregularly and vary from day to day. They are apt to occur also in patients whose muscular power is not of the best.

There results a confusion of terms in speaking of nystagmus, some using spontaneous nystagmus to indicate the movements observed without any effort on the part of the patient and some to indicate a nystagmus in the extreme of rotation but without any vestibular stimulation. Barré suggests that spontaneous nystagmus should be limited to that which is observed with the patient at rest, that the term for the nystagmus observed when the patient gazes in one direction or another be revealed nystagmus, and that rotatory or caloric or galvanic nystagmus be applied to those movements seen during the tests. Furthermore, rotatory nystagmus may denote either movement of the eye about its anteroposterior axis or that which is provoked by turning. Barré would term the former gyratory nystagmus and call the other nystagmus by rotation.

In examining for nystagmus, Barré tries to avoid artificial conditions so that he does not use opaque or strongly convex glasses, and does not have the patient close one eye. Moreover, in the caloric tests he does not even have the patient glance to one side or the other. He would insist on the slow phase of the nystagmus as the important vestibular element.

He also calls attention to convergence nystagmus, which is fairly frequent, which appears on one side and is characterized by sudden, short and infrequent jerks in the direction opposite the lesion.

In regard to the question of nystagmus in cerebellar diseases, Barré points out that there are irregular movements of the eye in cases of cerebellar disease, but that these do not properly coincide with the description of nystagmus, in that the oscillations are free, ample, smooth and irregular and that they recall dysmetric movements. This is a clinical complex, which occurs especially in combined vestibular and cerebellar lesions and deserves to be called a vestibular nystagmus of the cerebellar patient; true cerebellar nystagmus does not seem to exist.

In the second part of Barré's report, the instrumental tests are under consideration. He gives first place to the caloric tests of Bárány. Several positions are recommended for the study of caloric nystagmus but it is not important to impose these positions until after the douching is finished, so that the normal position of the subject at rest is chosen. Nystagmus is observed in this normal position and the head is then rotated, inclined and retracted into the so-called position no. 3 of Brüning. The forward flexion of 30 degrees does not seem to be particularly important, but the reversal of the lateral nystagmus and flexing the patient's head forward to 90 degrees seems important. The position no. 3 is favorable in noting the actions of the vertical canals. Although the appear-

ance of nystagmus may be earlier if the patient looks to one side or the other, the threshold is probably better observed and border line cases are more easily studied if the patient gazes directly ahead of him.

In regard to the time element, the quantity, and the temperature, there has been some disagreement, and it seems well that, in order to standardize the test, the temperature of the water should be 27 C., the amount necessary to provoke normal nystagmus from 50 to 70 cc., and the time from twenty to thirty seconds. The flow should be regulated to 100 cc. per minute. In cases of defective reaction, the quantity may be increased or the temperature lowered to 20 C., 10 C. or even melting ice. The temperature is particularly important.

The technics of Bárány, Hautant, and Kobrak are given; the author is rather impressed by Kobrak's method, which consists in allowing 5 cc. of water at 27 C. to flow slowly into the ear and waiting for a short time to see if nystagmus appears. This will give nystagmus in many normal persons, and in all that are hyperexcitable. In those whose labyrinth is less excitable, it should be increased by steps of 5 cc. each. This renders the test long, and sometimes with the small amounts of fluid the duration of the nystagmus is so short as to render impossible the performance of other tests. This is an unfair criticism, however, because the deviations of the body and extended arms appear before the nystagmus and last longer.

In actual practice, it seems well to start with Kobrak's method, if there is a possibility of an irrigated labyrinth, and to use Brüning's if the first does not give satisfactory results.

The production of nystagmus Barré believes due to vasomotor reflexes brought about by the action of temperature on the nerve endings of the posterior labyrinth. After the nystagmus has been observed, the patient is made to stand with his eyes closed, and any deviation is noted. The arms are also extended and their deviation noted. The pointing tests may be substituted by the extension of the arms, but there is no particular reason for doing so. Again, the static tests seem more important than the dynamic.

In rotating in the Bárány chair, it is classical to complete ten rounds in twenty seconds to examine the nystagmus, and ten turns in ten seconds to provoke movements of the limbs and trunk. This has been generally adopted. If vestibular hypersensibility is suspected, the author reduces the number to five turns. The rotation should be uniform and the cessation fairly gradual. The position of the head is extremely important. Six different rotatory tests should be employed, with the head in various positions, in order to isolate the various canals, and these positions should be noted with the form that the nystagmus takes. The duration of the postnystagmus should be noted, and, following the observation of nystagmus, movements of the extended arms and trunk complete the test. The symptoms that the patient presents should also be noted.

The electric tests are performed with electrodes on either temple, and the galvanic current is sent through with increasing force. From 2 to 8 milliamperes is sufficient to provoke lateral deviation of the head and a tendency to fall. When these reactions have been noted, the circuit is broken and the direction of the counter-reaction is noted. During the voltaic tests, vertigo, nausea, deglutition and vasomotor disorders may be noted. The counter-reaction noted before may be used with varied effects, to bring out stimulation, for while a person may resist the slow increase of the current, he finds it impossible to resist the counter-reaction on the break. The method of action of the electric current is probably different from that of other stimuli.

The third part of Barré's report is entitled, "General Discussion of Instrumental Tests, Their Mode of Action, and the Interpretation of the Provoked Reactions." An inexcitable vestibular system on one side should not be called dead until all tests have shown it so. It should be noted merely that there is caloric areflexia or rotatory areflexia. Caloric tests are useful on account of their strictly unilateral character. The rotatory tests act on both labyrinths,

but, in the cessation of rotations, particularly on the side opposite to that on which the subject is turned. The galvanic tests seem to act not only at the periphery, but also on the central portions of the vestibular apparatus.

Barré believes that chilling or warming the depth of a labyrinth produces vasomotor reflexes. There is a constriction on cooling; vasodilatation on warming. He contends that this reflex will bring about a difference in volume and pressure of vessels which surround the membranous labyrinth and would provoke a general complete excitation. The differences brought about by changing the position of the head he believes are explained by the fact that various portions are made anemic or are congested, and that changes in the hydraulics of the labyrinth are thus brought about. (This statement provoked adverse discussion, on account of the belief that the endolymph was caused to flow within the labyrinth after the manner of a thermosiphon, and that this would more easily explain not only the opposite reactions to cold and heat but also the change in the nystagmus as the result of changes in the position of the head.)

The rotatory tests apparently act by setting up movement in the endolymph. The electric tests probably excite the peripheral endings of the vestibular nerves, are possibly cataphoretic in their action, and in this respect there is a strong analogy with the optic nerve, for a galvanic current sent through the forehead may provoke luminous impressions. Rotation excites the positive pole (the virtual negative pole), and at the cessation of rotation toward one side the labyrinth on this side is stimulated. The association of phenomena of irritation or paralysis is often seen. Vertigo is probably due to stimulation, and it may be seen in cases that present also diminished or absent reflexes. As for the diagnosis of multiple lesions, this is still in the future, and the question of otoliths is for the future to determine.

At the end of the report, Barré gives a series of comments which he uses with his pupils to guide them in the pathways of truth. They are worth noting:

1. Thou shalt not accept too quickly, too completely, or too long, current schematic ideas.

2. Thou shalt be objective, telling thyself that the patient is always right and that he presents facts which thou shalt first take down, even without understanding, in order later to obtain an idea.

3. Thou shalt employ only the terms which express exactly the facts which they indicate or signify.

4. Thou shalt accustom thyself to terminate long reflections by saying that thou hast not understood and that thou canst not conclude.

5. Thou shalt proceed by stages and shalt not engage in the study of a particular point when thou lackest adequate comprehension of the whole of the subject.

In discussion, Professor Rimini stated that Kobrak's method was useful because it could be used at the bedside of the patient and that the latent period gave a better idea of the excitability of the labyrinth than any other test with which he was familiar.

Professor Baglioni called attention to the necessity of considering the voluntary action of the patient in such tests as past-pointing, in extended arms, and that even the eyes were under certain control, and that the psychic factors should be taken into consideration even in the evaluation of the nystagmus.

Ferreri raised two objections, saying that in the first place the tests reveal not only the condition of the vestibular apparatus, but also that of the subcortical cerebellar centers. He also objected to Barré's opinion of caloric excitation, taking the stand that the endolymphatic current set up in the labyrinth would explain more easily the symptoms observed.

Torrigiani stated that unless the patient was caused to look to the side, nystagmus was apt to remain latent and that in this way only the eyes crossed by nystagmus become evident.

Ayala asked how the author would exclude the cerebellum from consideration since true nystagmus is observed in cases in which the labyrinthine area has not been disturbed.

Mingazzini also called attention to the necessity of delicacy in tests such as the Romberg to bring out incipient disorders. The pathway influenced by the labyrinth and provoking past-pointing is probably extrapyramidal, although this tells us little.

Bárány stated that the vestibular Romberg could easily be differentiated from the cerebellar Romberg sign in that, in the first instance, the head definitely preceded the body in its displacement, and that the cerebellar Romberg sign was not modified by the position of the head. He demonstrated his method of performing the Romberg test, keeping the patient standing with his feet together and pushing him this way and that by the shoulders in order to estimate the resistance made by the patient.

Rimini called attention to the difference between labyrinthine and cerebellar nystagmus, saying that the former was intense at the beginning and gradually died out, whereas the cerebellar nystagmus lasted longer and got progressively worse.

Barré in his reply insisted that in order that true nystagmus shall be present the labyrinthine pathways in the cerebellum must be involved. He agrees that the Romberg test should be made sufficiently delicate to pick up incipient cases, yet he doubts the efficacy of too great refinement since more and more normals are apt to be included in the positives. Moreover, in the case of past-pointing, he does not believe that the deviation of the arms is a cerebellar sign since no similar reaction can be obtained by cooling the cerebellar cortex. He believes that the deiterospinal tract has more to do with past-pointing than the rubrospinal tract. He does not believe that voluntary actions in untrained persons can successfully simulate or prevent past-pointing. In hunting for nystagmus, Barré does not observe the lateral position of the eyes, but endeavors first to disclose nystagmus before the eyes are deviated. He holds to the point of view that nystagmus is truly vestibular and that a certain character assumed in cases of cerebellar lesion may be striking, but is nevertheless only a vestibular nystagmus in the cerebellar patient. When the labyrinth is dead to caloric or rotatory rotation, nystagmus may still be observed, and in these cases the galvanic tests are useful.

Although Barré accords the Kobrak test first place, he does not omit other tests when they seem necessary.

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THE SIGNIFICANCE OF INFECTION IN THE CLINICAL FEATURES AND PATHOLOGY OF DIFFUSE SCLEROSIS. FRANZ BIELSCHOWSKY, J. f. Psychol. u. Neurol. **33**:12, 1927.

A man with a family history of nervousness, except for a history of inflammation of the middle ear in adolescence, was never ill until the age of 50 when all his teeth came out for no discoverable reason. At 53 he began to have attacks of fever ranging as high as 39 C. (102.2 F.) without evidence of involvement of any particular organ or system. These febrile attacks were followed by impotentia coeundi and general prostration. During four years he had three febrile attacks, the afebrile periods between them lasting twenty-one days. During the third attack there appeared for the first time definite signs of involvement of the nervous system—apraxic-aphasic phenomena and evidences of left-sided involvement of the pyramidal tract; with the subsidence of the fever, on the tenth day after the onset of the attack, the symptoms referable to the nervous system also disappeared. Three weeks later, after a two day prodromal period, the fever recurred, the temperature rising to 39 C.; it was associated with symptoms pointing to extensive cerebral disease. As the entire history was that of a chronic recurrent infection, a blood culture was taken; it proved negative. After eight days of fever, the neurologic

symptoms had abated and the general condition again became normal. After several weeks of apparent health, the patient developed epileptiform seizures which seemed to have begun in the left arm; with this, the fever recurred, rising again to 39 C., and the neurologic symptoms returned. All attempts to determine the nature of the infectious agent were futile. He again made a complete recovery, but several weeks later the fever and neurologic manifestations recurred. Lewy, who saw him at that time, diagnosed atypical multiple sclerosis, and Simmons thought that the patient was suffering from "late epilepsy." The patient had six such attacks. During the last one, a diagnosis of cerebral neoplasm was made, but owing to the impossibility of localization, no operative procedure was undertaken. The man soon became comatose and died three days after the onset of the attack.

Histologic examination revealed: (1) a diffuse pathologic process characterized by demyelinization of the cortex of both hemispheres with well preserved axis cylinders; (2) cellular deposits of macrophages and lymphocytoid elements in the leptomeninges; (3) inflammatory vascular infiltrations in the meninges, cortex and spinal cord with regressive and progressive changes in the cells of the vessel walls; (4) perivascular glial foci within the white substance of the cortex; these foci were unusually marked in the frontal lobes, extending as far as the fifth and sixth cortical layers; in some areas the glial foci were also seen in the gray substance; (5) numerous fiber-forming glial elements in the demyelinated areas; (6) micro-organisms in the meninges and crebral cortex; (7) chronic disease of ths cells in the inferior olive. In view of these observations, the pathologic diagnosis was encephalitis periaxialis diffusa.

Schilder was the first to separate the clinicopathologic entity from the large group of diffuse sclerosis. He, also emphasized the exogenous inflammatory genesis of the disease. Regardless of the significance that may be attached to the bacterial manifestations in the case reported there is no doubt that the inflammatory reaction was due to some virus that had reached the brain through the blood channel. No one with an opportunity to examine the microscopic sections could raise the question of an endogenous degeneration or of a blastomatous process as described by Cassirer, Lewy and others. In addition to the inflammatory focal changes, there were also evidences of a degenerative process that must have been due to a toxic agent. This combination of inflammation and degeneration is found not only in encephalitis periaxialis diffusa but also in exogenous diseases of the central nervous system (e. g. dementia paralytica).

The author believes that the changes in the meninges and cortex in this case have not been reported before. The lesions in the leptomeninges and cortex bear a close resemblance to those described by Spielmeyer in typhus, the differences being that in Bielschowsky's case the regressive changes in the vessel walls were more pronounced and that there were evidences of more marked participation of the mesenchymatous cells in the reaction of the blood vessels to the virus. Another difference was seen in the radiation of the vessels into the cortex; whereas in typhus the infiltrations of the vessel walls cease at the cortex, in the case reported the infiltrations of the pial vessels extended into the cortex as they do in dementia paralytica. These differences, however, are not sufficient to raise any doubt as to the pathologic diagnosis of encephalitis periaxialis diffusa, because in all other respects the case fulfils all the characteristic features of the disease postulated by Schilder: extensive destruction of myelin with comparatively well preserved axis cylinders, arrest of the process at the cortex with preservation of the region of the U fibers, and inflammatory changes in the vessel walls. It must be admitted, however, that here and there, owing to an unusual acuteness and intensity of the process, the classic histologic picture is interrupted by regressive changes in the vessels leading to necrobiosis. It is also noteworthy that in this case, in contrast to most of those reported, both pyramidal tracts in the medulla were intact.

Clinically, the case was characterized by the symptomatology of a chronic recurrent infection: increase of monocytes in the blood, increase of bilirubin in the blood, and the presence of urobilin in the urine.

A review of the literature of diffuse sclerosis and its relation to infection reveals: In case 3 of Schilder, the first neurologic signs appeared during convalescence from a grippal infection one month before; the necropsy in this case showed a healed verrucous endocarditis of the mitral valve. A similar lesion in the aortic valve was found in the case reported by Braun. In Klarfeld's case (1922), the first neurologic signs appeared with an attack of grip; for weeks prior to death, the patient's temperature was 39.5 C. (103.1 F.) without demonstrable cause. Klarfeld includes this case among the purely degenerative forms of diffuse sclerosis, but he emphasizes the impossibility of making a sharp differentiation between the inflammatory and degenerative forms. Bielschowsky is also of the opinion that it is not always possible to draw definite conclusions from the anatomic picture as to the genesis of the disease. Cases showing high temperatures without demonstrable etiology are also reported by Krabbe, Ceni and Weichmann; Claude and Lhermitte also reported one which began with an attack of angina and ran a clinical course similar to the case reported in this communication.

Postmortem bacteriologic examination showed the presence of diplococci and streptococci. The micro-organisms were partly intracellular, and most of them were in the adventitia of the severely affected blood vessels. Although Bielschowsky does not commit himself definitely as to the precise relation of these bacteria to the pathogenesis of the disease, he is certain that they were due neither to contamination nor to agonal changes.

Almost every author who has studied diffuse sclerosis has included the inflammatory cases in the multiple sclerosis group. In 1906, Marburg proposed that the disease be designated encephalomyelitis periaxialis scleroticans because the acute cases were, in his opinion, proof that their genesis was inflammatory. Schilder, like Marburg, speaks of an encephalitis periaxialis; but Marburg emphasized its cicatricial character and added the adjective "scleroticans"; Schilder used the adjective "diffusa" to designate the localization of the process. There is, however, a more important difference between these two conditions — an essential pathologic difference; in encephalitis periaxialis there is, in addition to the process of demyelinization, a greater abundance of cells in the pathologic foci. The so-called "Markflecken" commonly found in multiple sclerosis and dementia paralytica never occur in encephalitis periaxialis diffusa. The "patchy" loss of myelin in the cortex in multiple sclerosis corresponds to the increase in granular cells found in encephalitis periaxialis diffusa. The degenerative changes of the vessels observed in encephalitis periaxialis diffusa are never seen in multiple sclerosis. The two diseases also differ in the nature of their scar formation: in multiple sclerosis there is hyperplasia of the glial network; in encephalitis periaxialis, the increase in glial fibers is relatively slight. This can readily be detected by examining the consistency of the brain: the sclerotic plaques can be distinguished from the adjacent tissues by their hardness; the demyelinated regions in encephalitic periaxialis diffusa feel much softer.

The two conditions have resemblances: (1) lymphocytes and plasma cells are encountered in both owing to the more or less prolonged duration of the inflammatory process in both diseases; (2) in both, the axis cylinders are more resistant than the myelin sheaths, and healing occurs in both by the formation of glial scars. These characteristics of nonpurulent inflammations of the central nervous system indicate uniformity of pathogenesis even though they offer no clue as to etiology. Diseases of different etiology may anatomically resemble one another closely; the same etiologic factor also may give rise to entirely different histologic features. This raises the questions: Are encephalitis periaxialis diffusa and encephalitis periaxialis scleroticans as different from one another etiologically as a tubercle from a gumma? Are

the former as closely related to one another as is an exudative phthisis involving an entire lobe of one lung to a patchy tuberculous peribronchitis? Bielschowsky selected these examples because cheesy pneumonia can occur only when, in addition to tubercle bacilli, the lung is invaded also by streptococci and staphylococci. The presence of various bacteria at necropsy in the case reported might confirm the suggestion of Braun that before the pathologic picture of diffuse sclerosis is developed, there must be added to the virus of encephalomyelitis "something else," perhaps some bacteria.

The close relationship between encephalitis and multiple sclerosis has also been emphasized by Jakob who in one case found sclerotic plaques adjacent to foci of typical Schilder's disease. Whether, as Jakob suggested, there are differences in the quantity of the deleterious agents, or whether endogenous or exogenous factors give rise to the differences in the process, are questions that cannot be answered until the etiology of the disease has been discovered.

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ENCEPHALOCELE: THE ANATOMIC STUDY AND SURGICAL TREATMENT IN FOUR CASES. J. PIQUET and R. TRAMBLIN, Arch. franco-belges de chir. **29**:945, 1926.

Defects in closure of the neural tube at the cephalic extremity are divided anatomically into meningocele, encephalocele, encephalomeningocele and encephalocystocele, depending on the degree of prolapse of the intracranial contents. Most of these defects develop at the craniospinal junction, although some are found in the ethmoid region and may be even orbital or intranasal in location.

CASE 1.—A new-born infant had a tumor the size of a large nut at the posterior fontanel. It was covered by a thin integument sliding over the underlying parts. There was no escape of fluid. The pedicle was broad; normal skin covered most of it and was sharply limited by the presence of the cuticle. It was fluctuating but not reducible, showed no increase in size when the child cried, and pressure on it was not communicated to the anterior fontanel. Hydrocephalus was not present. On account of slight excoriation, immediate operation was decided on. The pedicle was dissected free from the overlying skin and cut with scissors. No fluid escaped, but the pedicle was ligated and the skin sutured. The dressing remained dry; the stitches were removed on the tenth day and primary union resulted. Two years later the child was in good health, and hydrocephalus was not present. Some intellectual deficit probably was present. Examination of the tissue did not disclose nerve elements, only a fused pia-arachnoid in immediate contact with the epidermis.

CASE 2.—The patient was seen at the age of 10 days, when he presented a tumor at the junction between the head and the neck as large as a hen's egg. It was fluctuating and reducible with difficulty, and became firmer when he cried. It was covered by a delicate transparent cuticle beneath which a whitish mass, resembling nerve tissue, glided under a layer of fluid. Fluid did not escape but a brownish scab was present in the posterior portion. The normal skin extended about 15 mm. up the broad pedicle and then thinned out quickly. The pedicle was sectioned circularly at the junction of normal skin and the thin epithelium. Cerebrospinal fluid escaped immediately and a grayish-white mass became visible, which was sectioned at the level of the occipital bone. The deep part of the pedicle slipped into the cranial cavity. It was ligated with catgut. The bony orifice was 1 cm. long and a few millimeters wide. A double tier of sutures was introduced, the first uniting the fibrous pericranium and the deep parts of the pedicle (comprising also probably the meninges) and the second, the superficial integument. A few days after the operation, signs of hydrocephalus developed. Four months later definite hydrocephalus, divergent strabismus and pupillary inequality were present, but the child seemed to see and the pupillary reflexes were normal.

Every four or five days from 100 to 150 cc. of cerebrospinal fluid was removed, and the cranial bones then fell together. The child bore these punctures well, and it is possible that the improvement will continue. Anatomically, the tumor was composed of a thick epidermis without papillae, the deep layer forming a straight line and not containing hair follicles or sweat glands except in the extreme lateral portion. Beneath this was a vascular connective tissue which thinned out at the summit, giving rise to a delicate edematous tissue. Beneath this layer, there was nothing that could be recognized as dura, this being absolutely lacking as is the rule in all the encephaloceles. Its place was taken by delicate connective tissue, rich in congested vessels, which was evidently the meninges and, beneath this, principally in the apical region irregular strips of nerve tissue. In this tissue there were no nerve cells, only glia cells with delicate parallel fibrils running in various directions. Occasional cells contained sufficient cytoplasm to be visible. The structure resembled the superficial layers of the cortex. On the extreme inner portion there were ependymal vestiges and a villous structure of connective tissue covered by cuboid epithelium, each villus containing ramifying blood vessels, the whole greatly resembling the structure of the choroid plexus. This specimen, therefore, seemed to represent the ectopic prolongation of the ventricular cavity.

Case 3.—The patient, aged 1 month, was in good health. The neck was unusually short, the head resting between the shoulders; otherwise, it was normally formed. At the junction between the head and the neck was a voluminous tumor, one half the size of a child's head, with a broad pedicle. No fissure was felt in the skull. The pedicle was covered with hair and the tumor with a thick epidermis thinning out toward the convexity but not transparent. It was soft, fluctuating, did not increase on crying, and pressure on it was not transmitted to the anterior fontanel. The cutaneous collar was dissected and the pedicle of the cyst opened. A large amount of fluid escaped and the pouch became flattened. A large cranial perforation was found at the base of the occiput in which the end of the finger could be inserted. An attempt was made to close this perforation by fibrous tissue reflected from the pericranium, but free hemorrhage resulted, which necessitated compression. There was sudden cessation of the bleeding and syncope occurred; in spite of artificial respiration death resulted. The histologic examination revealed thick normal skin in the region of the pedicle containing hair follicles and glands with a normal subcutaneous tissue in contact with a delicate fibrillar structure containing vessels, the arachnoid. Lining the cavity was a delicate layer of nerve tissue without nerve cells, formed by neuroglia fibrils and cells. The innermost portion was formed by flattened cells resembling endothelium, but probably of ependymal origin. At the apex of the tumor, the stratified squamous epithelium did not contain glands or follicles. The subcutaneous tissue was finer; the arachnoid and nerve tissue was the same as in other places. This cyst also was considered to communicate with the ventricles.

Case 4.—The patient, aged 14 months, had a tumor the size of a bantam's egg which was located anteriorly at the root of the nose almost in the median line and was bilobate. It was excessively mobile, the pedicle being thin. It fluctuated, swelled slightly during crying and was not reducible. The fissure was palpable at the root of the nose, and the nasal bones seemed separated. The right eye was covered by its lid, although the eyeball seemed normal. Incision was made about the pedicle, and the large cutaneous collar was preserved. The pedicle was further dissected, the escape of cerebrospinal fluid being avoided, and it was then ligated and cut. The cranial opening was surrounded by the frontal, ethmoid and nasal bones. The fibrous elements in the vicinity were used in the deep closure, and the skin was sutured. Shortly after the operation the temperature rose, the child cried incessantly and death resulted in twenty-four hours. Examination disclosed an irregular epidermis containing hair follicles and sebaceous and sweat glands, with a normal

underlying dermis. There was no sharp distinction between the connective tissue of the skin and the meninges, however, nor anything suggesting dura mater. In the central portions, large vessels appeared and a few striated muscle fibers. In the central portion islands of nerve tissue were found disposed about a central core without an intervening space. Nerve cells were not found in these areas, a neuroglia reticulum alone being present. Some giant neuroglia cells were revealed, but there was no cavity and nothing resembling ependymal cells.

Study of these four cases shows that there is a pure meningocele and a pure encephalomeningocele, but that most of the tumors contain ectopic ventricular prolongations and, occasionally, a definite choroid plexus. The type formed by a layer of ependymal cells immediately beneath the pia, as described by Muscatello, must be rare but its occurrence evidently is possible, judging by the forms found in this study.

The treatment of encephalocele has undergone improvement recently, although until early in this century operation was rarely practised. The operation is best performed as early as possible in order to avoid the advent of ulceration and infection. When the encephalomeningocele is large and associated with cranioschisis, operation is of no use; if there is serious malformation of the skull or a fissure involving the foramen magnum and the spine, operation should be refused. Large cranial openings, if operated on, usually terminate in hydrocephalus, and hydrocephalus itself is a contraindication. Only children in good general condition should be operated on, and in those showing manifest infection in the eyes, ears or skin, operation should be avoided.

The classic operation consists of dissection of the cutaneous coverings, ligation of the pedicle and puncture of the sac. This is followed by extirpation of the pedicle and reduction of the herniated cerebral tissue, the escape of cerebrospinal fluid being avoided, if possible; the cranial gap is closed by fibrous tissue of the pericranium before suture of the skin. The authors believe that it is not necessary to open the cyst or reduce the herniated parts of the brain. Drainage does not seem to be necessary. Escape of cerebrospinal fluid often leads to collapse and should be avoided if possible. A flap may be necessary to close the skin; it is sometimes more difficult to close the osseous breach. The danger of infection is not great if infection does not exist at the time of operation. Among the later results, hydrocephalus is unfortunately frequent but can be treated fairly well by repeated lumbar punctures.

FREEMAN, Washington, D. C.

A LIPOMA ON THE SPINAL CORD ASSOCIATED WITH HYDROSYRINGOMYELIA AND OTHER MALFORMATIONS. MAX BIELSCHOWSKY and BRUNO VALENTIN, J. f. Psychol. u. Neurol. **34**:225, 1927.

The subject of this report was an infant whose parents noted immediately after its birth that: the feet were held in a peculiar hatchet-like position; the child was hydrocephalic; it had an opacity of the lens, and malformation of the genitalia consisting of large labia majora with poorly developed labia minora and a fairly well developed penis between the latter. The roentgen-ray examination did not show spina bifida. The infant died of pneumonia at the age of 1. Necropsy revealed marked internal hydrocephalus, hatchet-shaped feet and pseudohermaphroditism. When the spinal dura was opened, the lumbar enlargement of the cord was unusually large and had on its dural aspect a tumor the size of a plum, which became thinner as it ascended toward the lower thoracic cord. The dural sac was completely closed in the region of the tumor and was nowhere adherent to the vertebral canal. Microscopically, the tumor was a pure lipoma adherent to the inner layer of the pia. The posterior roots of the lumbar cord were surrounded by the lipoma and were partly displaced ventrad. At the level of the tumor the cord itself was edematous; after fixation, its gray substance was found somewhat disorganized.

The lumbar segments of the cord presented macroscopically a cleft-like cavity along the posterior longitudinal septum; cephalad, this cavity was larger and assumed the shape of an equilateral triangle with the base in the posterior commissure and the apex almost at the periphery of the cord. The position and form of the cavity corresponded to that of a typical syringomyelia. The inner lining of the ventral portion of the cavity consisted of one layer of ependymal epithelium, whereas its middle and dorsal portions had no ependymal lining. The cavity wall consisted of sponge-like strips of glia with moderately well preserved nuclei. Here and there one could see short rows of nuclei and fiber-forming glial cells with relatively large plasma cells. Sections of the cord in the region of the cavity stained by the Weigert method showed well developed anterior horns with an approximately normal number of nerve fibers and ganglion cells. The motor ganglion cells were uniformly shrunken; in many of them the tigroid substance was barely recognizable and contained remarkably small nuclei; owing to the formation of cavities, there was marked displacement of the posterior horns. The anterior columns were well developed, but both lateral columns were unusually small; the posterior column was displaced posteriorly and was so distorted that it was impossible to recognize the dividing line between the columns of Goll and Burdach.

The entire picture was one of maldevelopment of the caudal portion of the cord. Although the lipoma was a typical fatty tumor, the pia in the region of the latter was markedly thickened, especially over the posterior and lateral surfaces of the cord; it formed a fibrous ring with prolongations into the substance of the anterior and lateral columns separating the nerve fibers within the cord itself. The authors had no doubt that there must exist a close genetic relationship between the lipoma and the fibrous formation of the pia; it was apparently a malformation of the leptomeninges that occurred in the process of the closure of the medullary tube at the time the cord and dura were united. It was then that mesodermal elements became fixed on the surface of the cord, later invading the substance of the cord itself. The striking aplasia of the posterior horns also bears evidence of the time of formation of the lipoma and represents a failure of organogenesis which must have occurred early. Normally, the posterior columns, in their structural relationship to the posterior nerve roots, are well advanced in development at the end of the second month of intra-uterine life; at this period their area is relatively rich in nerve fibers; certainly it is richer than in sections taken from the lipomatous region in this case. Since the topography of the changes points to a definite relationship between the lipomatous region and the dystopia of the nerve roots, one is led to assume that the lipoma was formed at a time when the centrally directed axons from the spinal ganglia either had not reached or had barely reached their respective segments.

The position and form of the syrinx in the segments of the cord proximal to the lipoma are typical of hydromyelia, but its wall bears all the characteristics of a marginal glial wall, such as is usually found in the vicinity of syringomyelic cavities. This would seem to confirm the views held by Bielschowsky, Henneberg and others that, ordinarily, there is no great difference between hydromyelia and syringomyelia, and that each of these conditions is more often due to a failure of development of the structures derived from the ependymal layers which contribute to the process of closure of the medullary tube.

The next question that arises is whether the closure of the medullary tube in this case was not inhibited by stasis of the cerebrospinal fluid. The marked internal hydrocephalus found at necropsy would be in favor of such a hypothesis. The fact, however, that the central canal in the dorsal and cervical segments was not dilated would seem to point against this hypothesis. It must also be emphasized that in the region of the lipoma there was no evidence of any pressure effect on the central canal. Hence, one must assume that the lipoma and hydrosyringomyelia are coordinate manifestations, both of them due to some extraordinary defect in the germ plasm.

Although it cannot be denied that cases of this kind bear some genetic relationship to spina bifida occulta, no such factors enter into the case under discussion. The assumption that only early disturbances in the anlage of the cord and meninges are to be taken into consideration in attempting to explain the malformations in this case finds support in two additional factors: the presence of ganglion cells at the points of exit and of entrance of the nerve roots, and the presence of neurinomatous formations in the root zones of the segments of the cord covered by the lipoma. The occurrence of spinal ganglion cells in the posterior roots and in the pial margin of the cord, with the accumulation of cells in the thin neurinomas in the course of the posterior roots, can only be harmonized with the "theory of displacement." The simultaneous occurrence of hydrosyringomyelia with the pathologic changes in the same segments of the cord would also seem to be of significance, especially as the ganglion cell tracts belong to the region of the posterior line of closure.

The microscopic size of the accumulations of the nuclei and lamellar bodies in the posterior roots is no reason to doubt their neurinomatous character. Their small size is of special interest because it indicates that structurally these tumors, in their purest form, occupy an intermediary position between malformation and neoplasm. Such early anlages of tumors have hitherto apparently not been observed.

The pseudohermaphroditism seems not to have a direct causal relation to the changes in the nervous system, although indirectly it offers additional evidence that in maldeveloped persons there is a tendency ab ovo to malformations not only in the central nervous system but also in other systems.

KESCHNER, New York.

THE DEFENSE REFLEXES AND THEIR SIGNIFICANCE. P. FLEISCHMANN, Arch. f. Psychiat. **80**:377 (April) 1927.

The phenomenon of the so-called defense reflexes was known to physiologists long before clinicians began to make use of it, and dates back as early as the days of Descartes. It is mentioned by clinicians of the first half of the nineteenth century, was discarded for a while, but was again considered more seriously at the end of the nineteenth century, after Babinski discovered the reflex known by his name. The reflex consists in a contraction of a whole system of muscles, and may occur in the form of one of two types: (1) flexion, (2) extension. The first is the more common and consists in a triple or quadruple flexion: the extremity undergoes flexion at the hip, knee, ankle (dorsiflexion of the foot), and at times at the metatarsophalangeal joints. In most cases the toes assume the Babinski position. At times this flexion is accompanied by an inward rotation and slight abduction of the whole extremity. Following the flexion there is a return to the previously extended position. The rate of speed in which the different components of the act occur is disputed, but the majority of observers (the author's observations included) describe it as follows: After a latent period of from 0.25 to 0.4 seconds there is a sudden flexion of all the joints involved. After the acme of this is reached the contraction fades slowly and the action of the antagonists causes a slow return to the previously occupied position. The muscles involved are therefore the tibialis anticus, tensor fasciae latae, sartorius, gracilis, semitendinosus, and extensor digitorum longus (innervated by the second lumbar to the first sacral segments).

In a small group of cases, the extension reflex occurs. To demonstrate this, one must flex the limb at the hip and knee. A stimulus then causes extension of the hip and knee joints, and dorsiflexion of the foot.

A so-called normal defense reflex must be differentiated from the pathologic, and this differentiation depends mostly on the point of application of the stimulus. When the sole of the foot is stimulated, the flexion reflex may be obtained in normal persons as well as in pathologic states. The defense reflex

indicative of organic disease of the central nervous system must be elicited by stimulation of the dorsal surface of the foot or any part of the lower extremity above it. The type of stimulus differs, the most effective being that of pain (pin prick, pinching, or pressure). Temperature and friction stimuli may also elicit the reflex; many patients complain of involuntary movements (which are really defense reflexes) caused by the friction or weight of bed clothes, sudden changes in temperature, etc. These reflexes must be differentiated from voluntary movements. The attention of the patient must be distracted while trying to elicit the reflex. The quantitative and qualitative components of the reflex are to be utilized in differentiating it from voluntary movements. The sharp flexion and the rather slow lytic release, the stereotypy of the movement on repeated stimulation, and comparison between the two sides in cases of one sided lesions, will suffice as differential points. These criteria will also help to differentiate this reflex from the contralateral defense reflex observed in general hyperalgetic conditions. In these cases it is especially the return to the previously occupied position that helps in the differentiation.

The relation of other pathologic reflexes to the defense reflex is disputed.

The Babinski Reflex: Babinski, and some other observers, believe that there is no relation between the two. This is supported by the fact that some cases may show the one without the other. Other observers think that the two phenomena are one and the same thing. The author thinks that, not being absolutely clear as to the mechanism behind these two processes, one cannot state definitely either the one or the other contention, but there is a definite relation between the two in that they mostly occur in the same types of lesions. Patients in whom a Babinski reflex is observed without the defense reflex, or vice versa, will, if repeatedly examined, show the other after a while.

The Oppenheim Reflex: Here, too, the same relationship exists between the two reflexes. The Gordon reflex, however, from the nature of the method whereby it is elicited and from its reaction appears to be part of the defense reflex.

The defense reflex has a definite place as a diagnostic criterion. The material in which the author was able to demonstrate this reflex consisted of fifty-seven cases (twenty-one multiple scleroses, seventeen hemiplegias, one pseudobulbar paresis, one thrombosis of the posterior inferior cerebellar artery, three cerebral syphilis with spastic phenomena, two amyotrophic lateral scleroses, three cord tumors, one cord compression, one spina bifida occulta, one syringomyelia, two familial spastic diplegias, two parapareses of unknown etiology, one case of Little's disease, one uremic coma). This material, as well as material reported in the literature, shows that the reflex is found in both cerebral and cord lesions, in one sided as well as in bilateral lesions. Although the most active forms of the reflex are found in cord lesions, they are just as certainly found in cerebral. The supposition of some authors (based on physiologic experiments on animals) that the defense reflex is particularly significant of total division of the cord from the brain, does not seem to hold true. The author comes to the conclusion that if there is any special type of lesion that is responsible for the occurrence of the defense reflex, it occurs in organic processes in which the pyramidal tracts are affected in some way. Nobody has yet obtained a true defense reflex with intact pyramidal tracts. From a diagnostic point of view, it is interesting to note that some authors recommend the utilization of this reflex in determining the level of cord lesions. It appears that in different cases (especially of cord lesions) the reflex can be elicited by stimuli applied to the lower extremities beginning with the dorsum of the foot and going upward to certain levels. Stimuli applied above that level do not elicit it. These authors believe that this level indicates the lower limits of the process. With the determination of sensory disturbances (anesthetic) as significant of the upper level, they thus determine the localization and extent of the lesion. These authors also believe that when this interval between the two levels thus established is very wide, the indications are those of an extradural lesion. When the levels are near each other one is dealing

with an intramedullary process. No definitely established theory has yet been formed to explain the mechanism of the occurrence of this reflex. The author, however, is inclined to agree with those who see a release of spinal reflex patterns from a superimposed brain control in the occurrence of defense reflexes in pathologic cases.

MALAMUD, Foxborough, Mass.

TUMORS OF THE FRONTAL LOBE. L. PUUSEPP, Fol. Neurop. Eston. **6**:150, 1926.

Puusepp observes that the operative removal of tumors of the frontal lobe gives less satisfactory results than removal of tumors in other regions on account of the fact that such tumors are usually diagnosed rather late and only when they have reached a large size. The localizing symptoms are rather atypical and are striking only in advanced cases. His presentation enumerates sixteen tumors and one localized hemorrhage. Two of these were endotheliomas and lay superficially. Most of them were gliomas or sarcomas and occurred in early adult life. Trauma does not seem to play a significant rôle.

In many cases the diagnosis is made difficult by the presence of signs of increased intracranial pressure, since the mental picture is distinctly clouded. Examination after the injection of hypertonic saline solution facilitates a topical diagnosis.

Psychic disorders are the most striking features, although there is loss of the sense of smell in almost all cases. Reduction in memory for recent events is the most constant psychic defect. Whether this is due to lack of attention or to loss of the power of fixation is unimportant. The attention is certainly reduced as can be brought out by having the patient subtract a series of 7's from 100; the patient soon forgets where he left off. There is no definite disturbance of intelligence, merely of attention and fixation. The intellect can be disturbed in cases of large tumors that have lasted a long time. Moria is disclosed, particularly in tumors of one frontal lobe compressing the opposite lobe, so that the whole frontal brain is disturbed. Moria consists in joking, restlessness, grimacing and humming, and "allerlei Dummheiten," childishness. When this passes, the patient goes back into an apathetic state. In cases in which the tumor lies deep in the frontal lobe, spatial disorientation is frequent and is especially marked when both hemispheres are compressed. It is apt to be of late onset. Somnolence, apathy and motionlessness are liable to be late symptoms. The patients have little insight into the severity of their illness and are often euphoric. Hallucinations and delusions are not so frequent as in dementia paralytica. Although disturbances in the moral sense are reported, the author has not observed these. In the terminal stage, the patients become incontinent. The smaller the tumor, and the more superficially located, the less marked are the symptoms. These symptoms are evidently different from those of simple increased intracranial pressure.

The physical manifestations are found in so-called frontal ataxia, in which there is serious disturbance of equilibrium with no loss of recognition of posture and the possibility of performing complicated movements. Occasionally, the head and eyes are turned toward the side of the tumor, and most of the patients show some nystagmus, which is of no localizing value. Disturbance of language is sometimes observed in left-sided tumors; and in those cases presenting right-sided tumors there is often a curious inability for the patient to speak aloud. Apraxia was observed by Puusepp in three cases, although one of these had associated astereognosis from an additional parietal tumor. Mimic was reduced, particularly in deep seated tumors and gave a masklike appearance to the face in some cases. The author refers this to possible damage to the nucleus caudatus. Tremor was likewise referable to such damage, and in two cases there was myoclonia of the opposite facial musculature.

Pyramidal symptoms were usually late, although weakness of the facial musculature and tongue was common. Exophthalmos was observed in two cases, and difficulty in swallowing in one case, although this was probably due to pressure. Anosmia was an important symptom on account of its frequency. In

the beginning, when only headache was present, the sense of smell seemed more acute than usual but became reduced on the side of the tumor during the course of the illness. Headache was an early symptom and tenderness was usually greater on the side of the tumor. The eyegrounds revealed papilledema and alterations of the retina in about half the cases, but in most instances these changes came late. Five patients had reduced vision, and seven of them had hemorrhages in the retina with dilated veins.

Albumin was increased in the cerebrospinal fluid, with normal or slight pleocytosis in many cases, and the sugar was high. Xanthochromia occurred in three patients with large gliomas.

The course of tumors of the frontal lobe is almost persistently downward, with acute exacerbations, probably due to hemorrhages, and occasional slight remissions. The differential diagnosis of tumor of the frontal lobe is difficult or impossible in the early stages, and one is forced to await the general symptoms. A diagnosis can be made, however, based on the psychic symptoms, and on the disturbance of equilibrium and of olfaction. The sequence of events with reference to general symptoms is important in this regard, and in such cases as those mentioned with intracranial hypertension, it is well to reduce the pressure before asserting an opinion. The roentgenogram occasionally is an aid. Puncture of the brain is sometimes useful, and ventriculography is occasionally indicated, but in cases of small tumors this method does not give a characteristic picture.

Finer differential features are possible in outstanding cases; thus, in conditions of the extreme frontal pole, weakness in memory and irritability are striking, whereas when the tumor is located deep in the substance of the lobe, ataxia, apathy, masklike features and tremor are more apt to occur. If the tumor is located farther posterior, pyramidal signs may appear, but the head and eyes may rotate toward the side of the tumor, and the speech may become whispered. Motor aphasia is observed when the left frontal lobe is affected. If the tumor lies between the lobes and compresses both of them, the psychic symptoms are outspoken and mental defect is apparent, equilibrium is severely affected, and unconsciousness may follow changes in position.

Puusepp performed eight radical removals, two partial removals and six decompressions. Three patients in the first group were well after three years, although five survived one year. In all these cases, the tumor was encapsulated and could easily be removed. The two-stage operation was performed twice, but recurrences developed soon afterward. Only one patient was reported improved a year after simple decompression. The operative result in cases of tumors of the frontal lobe depends on: (1) the early diagnosis of the tumor, since the smaller the tumor the better the result; (2) the encapsulation; (3) the position of the tumor, whether superficial or deep. Most of the tumors removed by Puusepp were the size of a billiard ball.

FREEMAN, Washington, D. C.

THE PATHOLOGY OF COMPRESSION OF THE SPINAL CORD. RYOJIRO FURUI, Arb. a. d. Neurol. Inst. a. d. Wien. Univ. **29**:169, 1927.

This contribution is based on a study of five patients: (1) A woman, aged 67, with caries of the eighth thoracic vertebra and pachymeningitis tuberculosa externa. The duration of the compression was more than six months. (2) A man, aged 42, with carcinomatous metastases in the vertebral column and gibbus of the lower thoracic spine. The duration of the compression was about ten months. (3) A man, aged 66, suffering from tuberculosis with compression of the midthoracic cord. The duration of the compression was six months. (4) Compression of the lower cervical cord. (5) Compression of the midthoracic cord. In all five cases, the changes at the site of compression varied according to the duration and intensity of the pathologic process. The more recent cases showed more defects at the periphery of the cord than the cases of longer duration. The outstanding pathologic process was a chronic

edema with disintegration of the myelin sheaths and axis cylinders. When the defects were not too extensive, the surrounding glia was intact, but in the cases showing large defects the glial roofs were torn and had apparently been resorbed; this gave rise to large, irregular defects with glial rests within them; the defects were full of lightly stained, often barely recognized, granular cells. The defect formations were more marked in the lateral and ventral columns of the cord than in the posterior columns and diminished gradually in size as they approached the center of the cord. It was striking that even though the compression was in the dorsal region the defect in the cord was more marked in the lateral and ventral than in the posterior columns.

The areas in the immediate neighborhood of the defects showed marked degeneration of the myelin sheaths. The degeneration appeared in two forms: In the first, it was characterized by swelling and lamellar loss of substance; it was found at the site of compression and occasionally above and below it, but more frequently and more markedly above it. In the second form, the myelin sheaths appeared on cross-section as if made up of individual, small, sharply outlined globules; in other areas these globules appeared unusually large and much paler in color. This form of degeneration was also found above and below the site of the compression. The most striking changes were found in the axis cylinders. These changes were not at all like those usually observed in inflammatory processes of the cord. Although on longitudinal section the axis cylinders appeared wider and thicker at the periphery than in the center, they were not as swollen as in myelitis; the more delicate axis cylinders appeared less swollen but more lacerated, irregular and spiral shaped; this was especially the case at the site of the compression.

The author is unable to evaluate the changes in the vessels because his patients were all more than 60 years of age and presented marked evidences of advanced general arteriosclerosis. Among the tuberculous cases were some in which the vessels did not show the slightest evidence of inflammation—not even glial irritation. Occasionally, however, one could see some granular cells in the lymph spaces around the vessels at the site of compression.

The number of cases studied was too small to warrant definite conclusions as to the changes in the ganglion cells at the site of compression. All that could be said was that where the inflammatory process was most marked axonal degeneration could be observed; in areas in which there was no inflammation, two varieties of changes were noted: (1) loss of tigroid substance from the periphery of the cell; (2) loss of tigroid substance at the periphery with a marked increase in the center. In some cells the tigroid substance had disappeared entirely, leaving only the nucleus in the center; in some cases, the cells and their dendrons appeared swollen, with loss of tigroid substance at the periphery and its reduction to a fine powder in the center with retention of the nucleus. At the site of compression there seemed to have been an increase in the size of the cells. The cell changes described were most marked at the site of compression but could also be observed above and, to a lesser extent, below the compression. It must be emphasized, however, that not all cells seen on cross-section showed the changes enumerated.

In addition to the changes in the tigroid substance, there were also changes characteristic of the senium — serpiginous dendrites and lipoidosis. In the Bielschowsky preparation, there were no apparent changes in the fibrils; this, however, may have been due to the age of the material and its unusually long preservation in formaldehyde.

In the cases in which the pathologic process was old and far advanced, there was a dense glial sclerosis.

Generally, it may be said that the clinical manifestations were parallel to the anatomic manifestations, although now and then the anatomic symptoms were less marked than one would expect from the clinical picture. This, however, is not of special significance, because when compression gives rise to a functional disturbance it is impossible to say whether the latter is not due to

edema secondary to the compression. Furui does not consider the parenchymatous changes especially characteristic of compression of the cord, because the changes in the cells were so various that it is questionable whether some features of the pathologic process were not due to complications (in tuberculosis, to inflammation, and in persons more advanced in age, to senium).

KESCHNER, New York.

THE COLUMNAR ARRANGEMENT OF THE PRIMARY AFFERENT CENTERS IN THE BRAIN STEM OF MAN. WALTER FREEMAN, J. Nerv. & Ment. Dis. **65**:1, 149, 282 and 378, 1927.

The author attempts to define the primary afferent centers in the brain stem in terms of the known afferent centers in the spinal cord. As a preliminary study he shows that each mixed nerve, fifth, seventh, ninth and tenth, of the brain stem resembles the spinal nerve in carrying impulses from a certain cutaneous area, a certain portion of mucous membrane and a certain muscle group. He then shows how the opening out of the central canal into the rhomboid fossa displaces the entering roots to the ventrolateral surface of the medulla oblongata, but that the relationship between afferent and efferent portions remains the same, and indeed that the different fasciculi of the afferent roots maintain the same relative position that they do in the spinal cord. Direct segmental reflex fibers were particularly evident in the facial nerve, entering the motor root at the knee.

The primary afferent centers are then considered in detail. The spinal root of the trigeminal nerve is homologized with the tract of Lissauer in the spinal cord. The long caudal extension is expained as follows: The trigeminal nerve has usurped cutaneous fields formerly supplied by other nerves. The origins of the secondary pathways in the brain stem are laid down at an early period, however, and do not change their position. Therefore, in order to connect with the cells of origin of the secondary pathway, the nerve fibers coming through the trigeminus instead of through other nerves must travel a longer intramedullary course. The substantia gelatinosa probably transmits common cutaneous impulses for touch, pain and temperature, although tactile sensibility is probably but slightly represented.

The primary afferent centers for the relay of discriminative tactile and cognitive proprioceptive sensibility from the body are found in the nuclei of the dorsal funiculi. A nucleus of similar structure transmits stimuli coming through the trigeminal nerve. The author describes other nuclei lying in the same relative position with respect to the corpus restiforme which are probably concerned with the transmission of stimuli from the seventh, ninth and tenth nerves. The sensory nucleus of the facial nerve was found well developed in the elephant (appendix 1).

The relay centers for mucosal sensibility are found in the tractus solitarius, a continuation of the pars intermedia of the spinal cord. This tract is well developed in the three months' embryo and is recognizable as far cephalad as the entrance of the trigeminus. In the latter location, however, it deviates from its usual internal location and lies external to the proprioceptive area. The explanation for this may lie in the later development of the mouth, the centers that control structures of more recent development tending to lie farther from the central canal. Two nuclei lying close to the fasciculus solitarius and at the level of entry of the facial and glossopharyngeal nerves are considered to be the gustatory nuclei. In the fetal specimens examined by the author with his special silver impregnation method, those nuclei were evident. They can be identified however, in Weigert preparations of the adult brain.

Proprioceptive relay centers corresponding to Clarke's column are found in two locations, the descending and ascending roots of the vestibular nerve, and the mesencephalic root of the trigeminal nerve. The descending root can

be followed directly into Clarke's column, and it apparently receives fibers not only from the eighth nerve but from the ninth and tenth. In this connection the author expresses his belief, supported by some evidence, that the eighth nerve is merely a highly specialized proprioceptive division of the seventh nerve, and that it has usurped functions previously carried out by the fifth and tenth nerves. The long intramedullary course of the primary afferent fibers is explained on the same basis as that of the fifth nerve fibers.

The author accepts Johnston's thesis that the nucleus mesencephalicus trigemini comprises sensory ganglion cells that have remained intracerebral like those of amphioxus, and that these cells have to do with kinesthetic impressions from the muscles of mastication. This thesis is amplified, however, by the inclusion of the extra-ocular musculature. An adequate number of ganglion cells has not been disclosed to care for the large number of afferent fibers contained in the third, fourth and sixth nerves; the nucleus almost disappears between the fifth and fourth nuclear levels and then grows to large proportions at the level of the oculomotor nuclei; some fibers from the trochlear nerve were traced into this nucleus by Golgi; in a case of oculomotor paralysis studied by van Valkenburg, the nucleus was much reduced on the paralyzed side; finally, after exenteration of the orbit in the cat, the author noted rather striking reduction in the number of globoid cells on the operated side at the level of the third nucleus, while enucleations of the eyeball did not produce recognizable changes. Although the nucleus mesencephalicus trigemini seems to relay kinesthetic impressions from the muscles of mastication and from the extra-ocular muscles, these nerve cells are primary neurons with peripheral prolongations. The relay center corresponding directly with Clarke's column was not identified.

ALPERS, Philadelphia.

THE THERAPEUTIC VALUE OF MALARIA INOCULATIONS, MILK INJECTIONS AND SPECIFIC TREATMENT IN GENERAL PARALYSIS. I. SOMOGYI, Arch. f. Psychiat. **80**:312 (April) 1927.

This paper contains a comparative study of 400 cases of general paralysis. One hundred of these patients were treated with malaria, one hundred with milk injections, one hundred with specific reagents, and one hundred were not treated at all.

The Malaria Treated Cases: This treatment was begun in 1923. Most of the patients were inoculated with tertiary malaria, the plasmodia being obtained from nonsyphilitic patients. From 1 to 2 cc. of malarial blood was injected subcutaneously or intramuscularly (the author prefers the intramuscular injection and thinks it is even better than the intravenous). The conditions considered by the author as contraindications to malarial treatment were those of marked cardiac complications (advanced aortitis, myocardial degeneration, etc.), liver diseases, advanced tuberculosis, and other pulmonary diseases. The malarial infection was interrupted at the end of the course by quinine (generally quinine sulphate). The results are reported in terms of three groups. Group 1 comprises all patients who showed the best remissions and could return to their previous work. There were eighteen in this group. Group 2 comprises the cases that showed a remission, but in which some acquired defect was still present and the patients could not be considered as having reached their previous level. There were eighteen of these cases. Group 3 comprises the cases in which there was no change, a turn to the worse, or death. There were sixty-four in this group.

A study of reports by other authors and a comparison of these with his own results show quite a variation. Complete remissions (the author's group 1) range from 3.7 (Jansen and Hutter) to 50 per cent (Reese and Peter). The author believes that these variations depend mainly on the criteria used in determining the occurrence of a complete remission. Generally, the average runs from 25 to 30 per cent. Another source of variation lies in the type of material. In university clinics with material of an acute type, the results are

much better than in hospitals for chronic cases. The prognosis with this form of treatment is directly dependent on the duration of the attack (the best returns are obtained within the first few months) and the type of the disease (the best results are obtained with the acute, expansive forms and in taboparalysis).

Milk Injection Treatment: This treatment was carried out mostly before the beginning of the malaria treatment, that is, before 1923. Since then, milk injections had been given in cases that presented contraindications to malaria. The method consisted in injecting 10 cc. of carefully sterilized and filtered cow's milk into the gluteal region. From ten to twelve injections were given at from four to six day intervals. The temperature ranged between 102 and 105 F. The results in these cases were: group 1, twenty cases; group 2, twenty cases; group 3, sixty cases. Here too the best results were obtained with cases in which the disease was of short duration and of expansive and taboparalytic types.

Treatment with Different Forms of Specific Reagents: Different types of mercury, bismuth and arsenic preparations were used. The bismuth preparations have replaced the mercurial ones, and were found to give better results because they were not as prone to give rise to the untoward effects obtained with mercury. All were given by the intramuscular and intravenous methods: The Swift-Ellis, intracarotid and intraventricular methods were not used because there seemed to be no advantages to offset the greater risk. The results in a hundred cases were as follows; group 1, seven cases; group 2, ten cases; group 3, eighty-three cases.

Untreated Cases: These were patients admitted during the war when the adverse conditions at the clinic made it necessary to restrict treatment to part of the material only. Of these patients only five showed complete remissions (group 1), nine incomplete remissions (group 2), and eighty-six belong to group 3.

Comparison of the four groups shows they can be divided into two classes. The first consists of the 200 patients treated with malaria and milk injections, with 36 and 40 per cent remissions respectively. The second consists of patients treated with antisyphilitic remedies and those not treated at all. Here one finds 17 and 14 per cent remissions, respectively. The author concludes: (1) that the value of nonspecific treatment (malaria and milk injections) of general paralysis is established beyond doubt; (2) the specific (mercury, bismuth and arsenic) treatment seems to offer no better results than does no treatment at all. He believes that the value of the nonspecific treatment is in some way related to the high fever produced. As a proof of this, he considers the fact that no other common factor can be found between malaria inoculation and milk injection. He thinks that in this type of treatment the good results depend on the influence this treatment has on the inflammatory mysenchymal components.

MALAMUD, Foxborough, Mass.

THE PATHOLOGY OF THE CEREBRAL CORTEX IN IDIOCY. CHIKAZO INABA, Arb. a. d. Neurol. Inst. a. d. Wien. Univ. **29**:70, 1927.

The brains of two idiots were examined and in both the cerebellum was found larger than normal; the Purkinje cells were markedly diminished in number and in Nissl preparations not a single normal cell could be found; most of the cells were atrophied, and others showed ballooning in which the dendrites participated. The atrophic cells assumed grotesque shapes. The dentate nucleus was unusually well developed, almost hypertrophied, and showed many more convolutions than normal; it was also relatively richer in cells. There were no evidences of sclerosis. The nerve fiber bundles appeared unusually sharp, although here and there some paler areas were observed. The molecular layer was completely discolored.

The cerebral cortex was much smaller than normal, this being much more marked in the frontal than in the occipital lobes. All cell layers seemed to

be affected, the inner one being the only one that appeared even approximately normal; the fourth layer was most affected, the number of cells being less than half the normal. The contrast between the second and fourth layers was striking, the poverty of cells being greatest in the second and least in the fourth. The cells themselves were definitely atrophic, small in size with loss of prolongations, and were changed in structure. If it had not been for the preservation of the nuclei, most of these pathologic cells would hardly be identified as functioning elements.

The nerve fibers were more involved than the cells. No tangential fibers were seen. Fiber radiation was indistinct in the sensory regions (acoustic, sensory and optic), and no heterotopies were seen. The pathologic process in the fibers extended from the peripheral to the central part of the brain. Although the diminution of the cellular and fibrous elements of the cortex was replaced by a relatively marked increase in glia, nevertheless the picture was not that of sclerosis. The diminution of the cellular elements gave rise to a diminution in the size of the convolutions which made the sulci appear wider and deeper.

Inaba believes that in idiocy the malformation or developmental disturbance does not affect the cerebrum and cerebellum alike, but that the cerebrum is affected primarily and the changes in the cerebellum are secondary. He believes that the brain has completely developed but has become apparently secondarily reduced. He supports his views by the appearance of the pia and of the cerebral vessels. The former shows nodular thickenings, which are not due to secondary dilatations but rather to primary antecedent inflammatory processes, the residua of which could be demonstrated in some areas. The vascular changes are not those noted in presenile conditions but resemble those observed in cerebral syphilis. This may account for the fact that many authors consider that syphilis is the etiologic factor in idiocy. Unfortunately, no good anamnesis was obtained in the cases reported, so that this question could not be definitely determined.

In the concluding part of the study is a description of a normal but relatively small brain from a small person of normal intelligence. The smallness of the brain was well compensated for by an increase in cells. The number of cells was actually higher than that given by Economo and Koskinas.

As a result of his study, Inaba concludes that the outer layers of the cerebral cortex are more essential than the inner for intelligence; also that it is chiefly the cells of the second and the external parts of the third layer, and the external Baillarger and tangential fibers that mediate the higher psychic functions. In general, this view corresponds with that expressed in the literature.

KESCHNER, New York.

GENERAL PARALYSIS AND MILIARY GUMMA. C. I. URECHIA and U. MIHALESCU, Arch. gen. di neurol. psychiat. **7**:405, 1926.

In 1906, Straüssler described the occurrence of miliary gummas or syphilitic nodules in the brains of general paralytic patients. Often, these nodules are in relationship with the blood vessels while at times they are independent of them. The central portion of the nodule constitutes a homogeneous necrotic mass with lymphocytes and occasionally giant cells. More externally, lymphocytes and rod cells are found and at the periphery reactive neuroglia cells are present. The blood vessels of the vicinity are largely infiltrated by lymphocytes. According to Straüssler the lymphocytic infiltration is the expression of a more acute process, while the plasma cell infiltration indicates a more chronic reaction. Following the work of Straüssler, Witte, Landsberger, Chaskel, Marinesco, Ranke, Fisher, Jakob and others have described a combination of lesions of general paralysis and cerebral syphilis. Jakob describes, besides the nodules mentioned by Straüssler (giant cells and central necrosis), a more diffuse infiltrative lesion in which plasma cells, lymphocytes and a

few polymorphonuclear and epitheloid cells are found. The surrounding neuroglia tissue is hypertrophic and may show mitotic and amitotic division. In the small nodules (unorganized nodules) Jakob found no connective tissue proliferation. Spirochetes were present in the infiltrations. More recently, Grütter, d'Hollander and Rubbens, Hermel and Weimann have reported new cases of general paralysis combined with miliary gumma. According to Jakob, 50 per cent of the cases of general paralysis show vascular gummatous lesions, and 20 per cent of them miliary gummas, figures which Spielmeyer believes to be high.

Urechia and Mihalescu distinguish two types of syphilitic lesions in brains in cases of general paralysis — *A* — the granuloma or miliary gumma, and *B* — the necrotic area. The granulomas are more frequently found in the cortex (middle and deep layers) are exceptional in the white matter, and are always closely related to the blood vessels, as shown by serial sections. The early stages of the nodule formation are represented by an infiltration with lymphocytes, plasma cells and occasionally of polymorphonuclear and epitheloid cells which takes place between two or three capillaries and gradually increases in intensity. Giant cells are found only in more advanced stages or in typical miliary gumma. Macrophages as well as giant plasma cells may be encountered. In the tissue of infiltration the cells show partly degenerative changes. The microglia is proliferated and the astrocytes show hyperplasia proportionate to the stage of the lesion. Collections of lymphocytes or plasma cells may form simple nodules or granulomas with no presence of connective tissue. Gumma formation may also be seem in the adventitia of the blood vessels.

The necrotic areas are probably the result of the action of the spirochetes on the tissue, as in their early formation they are represented by accumulations of spirochetes in which the center undergoes gradual necrosis. Later, lymphocytes, polymorphonuclears, plasma cells, microglia and phagocytic cells appear. At times, as Schobb has described, formations recalling true small abscesses are found, not surrounded by connective tissue nor by collars of plasma cells and rod cells. Transitory forms between the areas of necrosis and the small abscesses may be found. The nodules and gummas may undergo a process of cicatrization ending in a glia-connective scar tissue.

FERRARO, New York.

THE CHROMAPHIL TISSUE AND THE INTERRENAL BODIES OF ELASMOBRANCHS AND THE OCCURRENCE OF ADRENIN. BRENTON R. LUTZ and LELAND C. WYMAN, J. Exper. Zool. **47**:295 (May 5) 1927.

One of the theories of the function of the suprarenal cortex is that it forms epinephrine or a precursor. The two suprarenal representatives can be separated without contamination from one to the other in the four species of elasmobranchs studied, namely, *Squalus acanthias, Raja stabuliforus, Raja diaphanes* and *Raja erinacea.* The interrenal body of *Squalus* is rod-shaped and is embedded in the posterior part of the kidneys. The chromaphil tissue is contained in thirty-four small, paired structures and in a pair of larger structures, the gastric ganglia, which are connected with the stomach by nerve strands. The gastric ganglia consist of a lateral portion of chromaphil tissue and a medial portion of nerve tissue. The interrenal bodies of the skates lie along the ventromedial borders of the kidneys and are extremely variable with respect to size, shape and number. The chromaphil bodies of the skates consist of a series of small, irregularly paired bodies and of an anterior pair of elongated, club-shaped bodies. Occasionally, a chromaphil body was found embedded in the capsule of an interrenal body. The gastric ganglia of the skates are loosely attached to the median borders of the anterior chromaphil bodies. In *Raja diaphanes* and *Raja erinacea,* scattered chromaphil cells are sometimes found in the gastric ganglia, but, in *Raja stabuliforus,* groups of chromaphil cells are regularly found in the edge adjacent to the anterior chromaphil body. In all three species there are two or three pairs of smaller, accessory ganglia

posterior to the gastric ganglia. The gastric ganglion of elasmobranchs is considered to be a primitive collateral autonomic ganglion.

Ringer extracts of the interrenal bodies, the chromaphil bodies and the gastric ganglia were tested by the mydriatic method for the presence of a dilator substance. A negative test was obtained from the interrenal bodies of all four species, and a positive test was obtained from the chromaphil bodies. In the gastric ganglia, the results varied with the different species depending on the presence or absence of chromaphil tissue in these structures. Extracts of these ganglia from *Squalus* were positive for a dilator substance; from *Raja diaphanes* and *Raja erinacea*, negative, and from *Raja stabuliforus*, sometimes negative and sometimes positive. Extracts of the accessory gastric ganglia from *Raja stabuliforus* were sometimes positive and sometimes negative. Tests made of extracts of kidney tissue were negative. All physiologic tests were correlated with the presence or absence of chromaphil tissue as seen in microscopic sections stained with bichromate solutions. It is concluded that the interrenal bodies of elasmobranchs do not manufacture or store epinephrine. The homology of this gland with the cortical tissue of the suprarenal gland of higher vertebrates leads to the conclusion that cortical tissue does not make epinephrine. The theory that cortical tissue is in any way concerned with the production of epinephrine is considered invalid in the light of comparative morphology and physiology.

WYMAN, Boston.

A CASE OF DUAL PERSONALITY. JULIUS DONATH, J. f. Psychol. u. Neurol. **33**: 1, 1927.

A well built man, aged 31, a physician and journalist, was suffering from mild apical tuberculosis; he had a psychopathic and tuberculous heredity and was himself psychopathic. During the World War he had sustained a severe electric shock to the right upper extremity; immediately thereafter he developed paralysis of that limb. After prolonged observation this was found to be psychogenic. At about the same time he was also infected accidentally with malaria, which lasted three years. At the beginning, the malarial paroxysms were associated with delirium, confusion and marked motor restlessness. Gradually, these mental symptoms became accompanied by periods of clouding of consciousness that would last from one or two hours to twenty-one days; during these periods, he was able to follow his profession of journalism, earned money and wrote long, intelligent and coherent letters to his friends; he would also send long distance telephone messages and would travel long distances—on one occasion from Capri to Vienna. In the course of these journeys he would regain his former consciousness, and on arriving in a hotel would make natural inquiries as to where he was and as to the date. During one of the attacks of clouded consciousness, he became engaged to two different women, and on regaining consciousness had complete amnesia for these episodes. During the attacks of clouded consciousness, there was marked increase in the general psychic functioning, so that anything that he undertook was done much better and quicker than usual.

Relatives and close friends readily recognized the dreamy state by the peculiar, somewhat sleepy appearance, the peculiar station and gait, and the loud voice. It was also evident that during these attacks there was a loss of inhibition in the mode of expression and judgment, and that he was unusually extravagant. The exciting causes of the attacks were chiefly in the affective sphere (excitement, worry, sympathy). There was no question of malingering; in his normal state he frequently complained bitterly and attempted to hide his condition.

Donath is inclined to believe that a vasomotor irritation occurred which was followed by vasocontraction, so that the blood supply to the higher centers was shut off. The possible effect of purely nervous influences, however, cannot be entirely disregarded. In this connection one has to recall only the disturbance of consciousness during hypnosis when it is suggested to the patient

that he is falling asleep. Of special psychologic interest in cases of dual personality that actually lead to a double existence is the close resemblance of the subconscious state to consciousness; this resemblance is so great that sometimes the two conditions cannot be differentiated; their recognition becomes especially difficult in consideration of the capacity for higher psychic functioning during the stage of secondary personality. The medicolegal significance of these factors is evident.

KESCHNER, New York.

SUBEPENDYMAL GLIOSIS IN A CASE OF GENERAL PARALYSIS. A. PFANNER, Arch. gen. di neurol. e psychiat. **8**:1 (April) 1927.

The author describes, from the histologic point of view, two areas of subependymal gliosis in a case of general paralysis. The general structure of the area was that of a glia reaction in which the fibroglia and plasmatic glia were found at different stages of development. In addition, there were numerous rod cells and large neuroglia cells of the giant type with numerous nuclei surrounding a central mass of a more or less distinct cytoplasm. Blood vessels were scanty in the areas of the gliosis. The author considers that the neuroglial giant cells have little to do with the formation of fibers, but that they represent a degenerative form of neuroglia probably destined to a destructive process.

The histologic feature of the subependymal gliosis leads the author to review the debated question of the origin of the neuroglia. The fact that numerous rod cells were found in hyperplastic neuroglia tissue, where no blood vessels nor mesenchymal reaction was detected, leads the author to the belief that the rod cells must have the same origin as the other constituents of the glia tissue. Pfanner accepts the view of Hortega concerning the transformation of the microglia into gitter cells, pointing out that the property of transformation makes the microglia similar in its reaction to the histiocytes of the reticulo-endothelial system. On the other hand, however, Pfanner points out that the formation of astrocytic giant cells is analogous to the formation of giant cells of mesodermic origin. Another analogy between the reaction of the mesodermic tissue and of the neuroglia tissue is the formation of perivascular nodules (especially in malaria, in typhoid, and in Chagas' disease). According to Dürck, the glia perivascular nodules may be termed granulomas in analogy with those of mesodermic origin. As a fact, Dürck reports that the tuberculous granuloma, at its earlier stage, is exclusively formed by glia cells which later are transformed into giant cells, giving to the nodule its peculiar mesodermic appearance (tuberculous granuloma). Dürck believes that the glial perivascular nodules are an expression of the organism's defense, a component of the inflammatory process, and that in the central nervous system the organism may utilize the neuroglia cells to build up the epithelioid cells of the granuloma.

These facts suggest to Pfanner the mesodermic origin of all neuroglia cells (astrocytes and microglia) as these cells react in the same way as the elements of the reticulo-endothelial system, the astrocytes corresponding to the fibroblasts and the microglia corresponding to the hemohistioblasts. The mesodermic origin of all neuroglia cells has also been considered recently by Rezza in his studies on the reticulo-endothelial system and on the Rio Hortego cells.

FERRARO, New York.

THE CENTRAL MECHANISM OF THE LABYRINTHINE RIGHTING REFLEXES: THE PHYSIOLOGY OF THE POSTERIOR LONGITUDINAL BUNDLE AND VESTIBULAR NUCLEI. SHUGORO NISHIO, Arb. a. d. Neurol. Inst. a. d. Wien. Univ. **29**: 60, 1927.

An injury involving almost exclusively the left posterior longitudinal bundle caudad to the abducens nucleus, in a rabbit, was followed by forced posturing of the head and forced movements to the side of the lesion with inability to bring the head back from the left side to its normal position when the animal

was blindfolded; the slight inability to rotate the head from the right side to its normal position rapidly disappeared. This type of lesion was also followed by an initial disturbance of the optical righting reflex (retention of abnormal movements of the head when the animal was allowed to keep its eyes open); this result was apparently due to the effects of shock.

Control experiments, during which the fourth ventricle was exposed and the superficial portion of its floor traumatized without injuring the posterior longitudinal bundle, were not followed by disturbance of the labyrinthine righting reflex. In these experiments, care was also taken to make the injury to the cerebellum minimal, although it had already been shown that ablation of the cerebellum had no effect on these reflexes.

These two sets of experiments, therefore, left no doubt that the disturbances of the labyrinthine righting reflexes were due to a lesion involving only the system of the posterior longitudinal bundle.

The next question considered was whether loss of the labyrinthine righting reflexes after injury to the posterior longitudinal blundle is due to section of the centripetal or the centrifugal fibers. The fact that the centrifugal component of the reflex arc (Rademaker) is in the rubrospinal tract, which is situated far from the point of injury to the posterior longitudinal bundle (a superficial lesion limited to the midline will not involve the rubrospinal tract), leads to the conclusion that injuring the posterior longitudinal bundle (as in the author's experiments) does not affect the centripetal component of the reflex arc. If, then, it is true that a lesion involving the centripetal portion of the reflex arc behind the abducens nucleus abolishes the labyrinthine righting reflexes, it is not likely that the fibers of the posterior longitudinal bundle originating in the vestibular nuclei anterior to the level of this lesion are of much significance in the formation of the centripetal part of the reflex arc. In other words, it is not the oral cell group of the vestibular nucleus column (Bechterew's nucleus, Deiters' oral cells) but the caudal parts of this nuclear region (Deiters' nucleus at the point of transition of the cells of the spinal roots as well as the nuclear elements lying on the roots themselves) that mediate the labyrinthine righting reflexes.

KESCHNER, New York.

SYMMETRICAL CENTRAL CEREBRAL SCLEROSIS AND DIFFUSE PERIAXIAL ENCEPHALITIS. C. FOIX and J. MARIE, Presse méd. **25**:417 (April 2) 1927.

A case reported by P. Marie and Foix, in 1913, as central sclerosis, is grouped with two cases observed since that time, to make a chronic form of the condition of which Schilder's diffuse sclerosis (1912) is here considered an acute form. Outside the thirty cases of Schilder's type reported since by various observers, Foix and J. Marie are inclined to place the cases of leukoencephalitis (Claude and Lhermitte, Jumentié and Valière-Vialex) in a separate category. Foix' three cases are analyzed at length, and seven figures are included. His central sclerosis and Schilder's diffuse periaxial encephalitis are compared as follows: While the pathologic lesions of both exhibit an identical distribution, the first is grayish and the second yellowish; the second is generally either harder or softer than the first, and shows little or none of its definite contraction of the white matter. Most important of all is the microscopic appearance: the neuroglial growth in the second is of recent character, rich in cells, including some giant and multinuclear elements, and showing leukocytic perivascular infiltration and numerous granular corpuscles. The neuroglial tissue of the first (the central sclerosis) is in an advanced stage, rich in fibrils and showing sclerotic perivasculitis. The two seem to be different stages of the same pathologic process.

Clinically, chronic central cerebral sclerosis passes through three stages, acute attack, improvement and residuals. The attack consists in the rapid development, within a few days or weeks, of a spastic paralytic motor syndrome in which early and well marked contractures predominate from the outset.

This stage may be interrupted by convulsions followed by paralyses. Sensory disorders, rapidly developing blindness in particular, may sometimes be the first clinical signs. After the first stage has lasted several weeks or months, mild improvement in some of the signs is gradually effected. The final stage commences about twelve or eighteen months after the original onset, and is marked by the absence of further change.

Diffuse periaxial encephalitis — here renamed fatal subacute central sclerosis — is characterized in its commonest form by three types of symptoms, any one of which may appear first: visual (diminished vision, hemianopia, retinal congestion), motor (spastic monoparesis, hemiparesis, or quadriparesis, with or without aphasia) and psychic (apathy, sluggishness). The picture becomes temporarily fixed after three or four months, with symptoms of increased intracranial pressure; further progressive paralyses, contractures and mental deterioration lead to death about twelve or eighteen months after the onset.

HUDDLESON, New York.

REFLEXES IN APES. G. D. ARONOVITCH, J. Ment. & Nerv. Dis. **65**:457 (May) 1927.

Many reflex symptoms found in nervous syndromes are only manifestations of evolutional regression to a lower level or function. To determine the biologic nature of such reflexes, comparative physiologic data are necessary, and the author has made a study of the reflexes in twenty-three apes, including *Macacus rhesus*, baboon, mangabey, orang-utan and lemurs, varying in age between 2 and 4 years. Pupillary reactions to light and in convergence were found in all. The corneal reflex was also present; such reflexes as the supraorbital and nasopalpebral were easily obtained. In none of the apes, however, did the muscles of the belly contract when the relaxed abdominal wall was stroked, although the abdominal wall of the ape does not show any essentially structural difference from that of man. The periosteal reflex described by Guillain as the "medio-pubic" elicited by percussion of the symphysis pubis, with resulting contraction of the abdominal muscles and adductors of the thigh in man, is defective in apes, as far as the abdominal resistance is concerned. The deep tendo abdominalis reflex also can be obtained in the ape, and this points to the appearance of the abdominal reflexes with the human acquisition of the erect posture. Thus, the anthropoids are not yet orthograde creatures. In male apes, no scrotal contraction comparable to the cremasteric reflexes in man was elicited. Knee jerks were readily obtained, and sometimes crossed knee jerks; so also was the achilles jerk. The usual plantar reflex of man with flexion of all five toes was not obtained. The author adds that investigation was not easy in all apes, some being excitable and fearful, in which cases an acute hyperreflexia was observed.

HART, Greenwich, Conn.

STRIOPALLIDAL AND BULBAR SYMPTOMS IN EXOPHTHALMIC GOITER. H. KLEIN. Monatschr. f. Psychiat. u. Neurol. **65**:138 (June) 1927.

Klein calls attention to the frequent association of exophthalmic goiter with organic diseases of the central nervous system, especially those involving the striopallidal and bulbar regions. He reports a case in which the symptoms referable to the central nervous system ran roughly parallel to periods of remission and exacerbation of the exophthalmic goiter. Improvement of both symptom groups was marked after each of two operations (partial thyroidectomy). Eventually, certain of the symptoms of exophthalmic goiter disappeared (tachycardia) while others persisted (exophthalmos). The encephalopathic symptoms grew progressively worse. On the basis of this and other cases, as well as because of the relatively frequent finding of intracranial, especially bulbar, hemorrhages at autopsy, he believes that the central nervous lesions are the result of the toxin present in exophthalmic goiter.

SELLING, Portland, Ore.

CEREBRAL DISTURBANCES, ESPECIALLY OF THE FRONTAL LOBE FUNCTIONS, IN INFLAMMATORY CONDITIONS OF THE SINUSES. M. DUFOURMENTEL, J. d. neurol. et d. psychiat. **27**:233 (April) 1927.

The author mentions the fact that he has been among the first to emphasize the importance of sinusitis as an etiologic factor in headaches, and then he calls attention to certain cerebral disturbances following some of the nasal diseases. These symptoms are mainly a diminution of memory, especially for recent events, which diminution results, on deeper analysis, as the attribute of a lack of attention, the patients being incapable of this as long as formerly. Another result of the lack of attention is the weakening of affect, which gradually is followed by depression. The symptoms, according to the author, are usually found in chronic inflammatory conditions of the nose or of the sinuses; in acute cases the focal phenomena presumably dominate the picture. Lesions of the ethmoid and frontal sinuses are especially liable to be responsible for the mental signs mentioned, which are homologized by the author to the symptomatology reported by Bianchi in his experimental work on the frontal lobes.

FERRARO, New York.

PERIODIC ATTACKS OF SLEEP FOLLOWING EPIDEMIC ENCEPHALITIS. DR. O. CAMPBELL, Monatschr. f. Psychiat. u. Neurol. **65**:58 (June) 1927.

The author reports a case of epidemic encephalitis characterized by alternating periods of sleep and of relative normality. The attacks developed five years after the acute phase of the disease. The sleeping spells last from two to three days. They begin with a sense of difficulty in initiating any movement. This can be fought off for a time, but finally overmasters the patient and he falls into a deep sleep. This differs from normal sleep in that he cannot be aroused. Muscle tone is increased, and queer and unusual postures are assumed. In the transition to the waking state, he goes through a phase of akinetic pseudosleep. During the free intervals, which also last two or three days, he shows mild parkinsonism. His sleep at these times is normal. Pulse and respiration are much slower during the sleeping attacks than during sleep in the free intervals.

SELLING, Portland, Ore.

SYNDROME OF MENTAL AUTOMATISM. PERCIVAL BAILEY, J. Nerv. & Ment. Dis. **65**:345 (April) 1927.

The syndrome of mental automatism, sensory, motor and ideoverbal, is regarded by Clérambault as due to a physicochemical alteration of cortical neurons and their connections, by a subtle and systematic fixation at an age when their resistance is greatest. This alteration may be due to infection, intoxication, degeneration or traumatism. The syndrome usually begins with the nonideational phenomena, echoes of thought, paresthesia and word salads. The subject, who is surprised by this phenomenon in a neutral affective state, usually gives them an exogenous explanation because of their strangeness and lack of connection with his habitual psychism and may react to them by erecting a system of ideas determined largely by his personality, the result being a chronic hallucinatory psychosis.

HART, Greenwich, Conn.

Society Transactions

SOCIÉTÉ DE NEUROLOGIE DE PARIS

Session of July 7, 1927

PROFESSOR G. ROUSSY, *in the Chair*

Rev. Neurol. **2**:52, 1927

TREATMENT OF GENERAL PARALYSIS BY INTRAVENOUS INJECTIONS OF VACCINE. SICARD, HAGUENAU and WALLICH.

It is sometimes difficult to determine how many malarial paroxysms a patient can stand; more than one patient has been allowed to continue beyond possibility of recovery. In searching for a safer method of producing fever the choice fell on the antichancroid bacterin of Nicolle. This is injected intravenously in doses beginning with from 250 to 300 million bacteria. Two or three hours after the injection, a chill occurs and lasts about one-half hour; at the end of that time, the "thermic apogee" is reached. The same dose seems to be effective in subsequent injections, since the patient does not develop tolerance. The temperature reaches 103 or 104 F. on each occasion. In some cases, even after forty injections, there seemed to be no damage to the kidneys or other organs. The clinical effects of this treatment seem to be comparable with those obtained by impaludation.

MALIGNANCY OF THE NEURO-EPITHELIOMAS. DRS. VINCENT and KREBBS.

Neuro-epitheliomas are considered benign and amenable to surgical treatment by some investigators, but certain of them are exactly the opposite. The diagnosis is made on the characteristic rosets, but the microscopic examination itself suffices to indicate a malignant condition. In the specimen shown, an enormous mass was present in the median portion of the right cerebral hemisphere, extending down and forming a hard, firm whitish mass around the aqueduct and reducing its lumen greatly. Sessile metastases were present in the cranial and spinal nerves. The invasive, proliferative and metastatic characters of the growth certainly indicate a malignant condition. Such neuro-epitheliomas appear sensitive to penetrating roentgen rays, but they recur in spite of enormous doses.

ARTERIAL ENCEPHALOGRAPHY IN THE LIVING. DR. EGAS MONIZ.

By means of the injection of sodium iodide, in 30 per cent solution into the internal carotid artery, the vascular tree of the brain is brought into sharp relief in the roentgenogram. This was carried out by the author, first on a cadaver, then on an experimental animal, and finally the technic was adapted to a patient. It was found impracticable to inject the solution blindly; in recent cases the artery has been exposed. The author advises the following technic: (1) prepare the patient with one or two injections of morphine and atropine; (2) expose the internal carotid; (3) fix the head of the patient on the roentgen-ray table so that movement will not take place; (4) puncture the carotid without allowing blood to enter the syringe; (5) avoid the entrance of air; (6) ligate the carotid temporarily; (7) inject immediately and rapidly from 5 to 6 cc. of sodium iodide (25 per cent) recently prepared and sterilized; (8) make one or more instantaneous exposures (as rapidly as possible), continuing to inject the opaque fluid; (9) remove the ligature

from the internal carotid. The roentgenograms are striking, and it is possible by visualizing the arteries and their deviations from the normal course to localize certain tumors.

Before the present technic was established, several attempts were unsuccessful. In case 1, the fluid was probably injected into the jugular vein; in case 2, the patient experienced pain and became violent; in case 3, the sensation provoked by the injection of strontium bromide was so disagreeable that further trial was abolished. A cervical sympathetic syndrome developed. In case 4, the fluid ran out into the deep cervical tissues and provoked fever but no abscess; the patient developed a typical Bernard-Horner syndrome. In three cases, the injections were made by the open method.

In case 5 (unlocalized tumor), the injection was not disagreeable to the patient, but it did not give results, since the time elapsed was probably too great. The patient had fever the next day and temporary dysphagia. In case 6, a parkinsonian subject, in which strontium bromide was injected partly as a trial for its direct action on the brain, the patient experienced severe pain, developed signs of thrombophlebitis and died forty-eight hours after the injection.

This experience finally led the author to discard the strontium salts and to use sodium iodide. This was done in three cases. In case 7 nothing was seen of the arterial tree, the substance having been swept out before the exposure could be made. The patient had some pain in the ear; the pulse rate dropped from 90 to 56; the patient had some disturbance in language; the temperature rose to 100, but was normal on the third day. In case 8, the patient was blind and the diagnosis was not made. The film was inconclusive, although the arterial distribution was not normal. The patient complained of mild pain in the temple, right eye and right ear. The pulse rate dropped from 95 to 60. There was some disturbance in language, but on the third day the man was normal. The last patient was a man with a tumor of the hypophysis, who presented a Fröhlich syndrome. The roentgenogram shows the carotid pushed forward and without its superior curve; the sylvian artery was also pulled forward and upward; the anterior cerebral artery was displaced and was thin and almost closed. The patient did not experience any particular effects from the injection.

NEW YORK NEUROLOGICAL SOCIETY

Regular Meeting, Oct. 4, 1927

LOUIS CASAMAJOR, M.D., *Vice-President, in the Chair*

COMPARATIVE STUDIES OF THE SURFACES OF ENDOCRANIAL CASTS OF MAN AND PREHISTORIC MAN. DR. ARTHUR WEIL.

A new apparatus has been devised for the measurement of endocranial casts. With its help the whole surface is subdivided in parallel stripes, the outlines and distances of which are measured and used for the calculation of the plane of these different stripes. Endocranial casts of the following skulls were investigated: normal man, Predmost, La Chapelle-aux-Saints, Rhodesian, Piltdown, Trinil, and, for comparison, of an adult male gibbon. The numbers found for the endocranial casts, corresponding approximately to cerebral and cerebellar surfaces, are given in table 1.

In measuring these parts of the casts, which correspond to the bones originally discovered, it was found that newly reconstructed surfaces were: in Piltdown, 53 per cent; Trinil, 39 per cent, Rhodesian, 14 per cent.

For the comparative study of parts corresponding to fossils, the cap of the cast only was measured. It was limited by a horizontal line connecting the

protuberantia occipitalis interna with the frontotemporal angle, and continued along the base of the frontal lobe. Three lines were erected perpendicular to this base line: (1) at the frontotemporal angle; (2) at the vertex of the posterior inferior angle of the temporal lobe; (3) at the cerebellotemporal angle. The cap of the endocranial casts was divided into four regions by these three lines. Measurements of the four divisions showed that the frontal part is most developed in man, and next in Piltdown. Region *2* is larger than *3* in man and Rhodesian, equal to *3* in Predmost, and smaller than *3* in the other casts. Table *2* gives absolute and percentage distributions.

Next, the part of the surface of the endocranial casts anterior to the first branch of the meningeal arteries was measured. The results, expressed in percentages of the total surface, were approximately the same for all casts. Outlines of sections through the casts in the three different planes were constructed with the help of wax rods. The largest diameters of such sections

TABLE 1.—*Numbers for Endocranial Casts of Skulls*

	Cerebral Surface		Difference Between l and r, in per Cent	Cerebellum, Sq. Cm.	Total, Sq. Cm.
	Left, Sq. Cm.	Right, Sq. Cm.			
Man	303	306	1	95	704
Predmost	327	327	0	104	758
La Chapelle-aux-Saints	337	339	0.6	86	762
Rhodesian	303	303	0	79	685
Piltdown	276	261	5.4	88	625
Trinil	224	225	0.4	78	527
Gibbon	42.6	42.9	0.7	19.3	105

TABLE 2.—*Absolute and Percentage Distributions in Measurement in Skulls*

	Left Regions of Cap of Cast in Sq. Cm.				Total Sq. Cm.	Percentage Distribution				Total in % of Cerebral Surface
	I	II	III	IV		I	II	III	IV	
Man	59.5	44.7	36.6	84.8	225.6	26.4	19.8	12.2	37.6	75.6
Predmost	55.0	50.8	53.1	84.7	243.6	22.6	20.9	21.8	34.8	74.5
La Chapelle-aux-Saints	52.4	45.7	47.4	100.8	246.3	21.3	18.6	19.2	40.8	69.9
Rhodesian	47.7	40.1	36.4	71.2	195.4	24.4	20.5	18.6	36.5	64.5
Piltdown	58.0	33.3	47.4	68.5	207.2	28.0	16.1	22.9	33.0	75.1
Trinil	35.7	32.3	37.8	52.4	158.1	22.6	20.4	23.9	33.1	70.7
Gibbon	6.2	5.1	9.2	7.0	27.5	22.6	18.5	33.4	25.5	64.6

were measured and compared. Horizontal sections showed a close resemblance between man and prehistoric men, while, in sagittal sections, the height was larger in man and Predmost than in the rest.

Finally, the different relations between different parts of the cap of the casts were expressed in percentages of the normal standard, 100, of modern man. The plus and minus deviations from the standard were represented in curves and the mean value with standard deviations was calculated. The sequence of approach to the modern standard was: Predmost, 108.32; Rhodesian, 110.0; Piltdown, 110.06; La Chapelle-aux-Saints, 111.62; Trinil, 116.30, and Gibbon, 126.10.

DISCUSSION

DR. LOUIS CASAMAJOR: Dr. Weil has added some more evidence of what primitive man was like. Much is known of his habits, especially from the implements of his life which he has left behind. The material must be analyzed to learn more about what kind of a fellow he was. Such work as this of Dr. Weil, and that which Dr. Tilney has previously published in regard to the brain of primitive man, is of great value, but it is difficult to draw conclusions. Dr. Weil has offered a method of approach to the visible cortex; what that

means in primitive man, in modern man, or in lower animals, remains for future determination. The angles formed by the lines and the relations between the various parts of the cortex are of interest; one would like to learn if these show any differences between different races and between mental defectives and the so-called normal man. Has Dr. Weil applied his methods of measurements to the brains of any of the lower animals with the view to determine whether there is any connection between the measurements and the conduct of the animal?

Dr. H. A. Riley: While Dr. Tilney was working on these intracranial casts I had the opportunity to study them. It always seemed to me that it was necessary to speak with the utmost conservatism in regard to any conclusions that might be drawn. Dr. Weil has spoken of the fragmentary character of the skulls which have been found; this shows well on the casts in which the original material contrasts with the part which has been supplied by man's art. Practically all one has on these casts are the markings of the meninges, which do not necessarily have any relationship to the underlying cortical geography. There is little indication of convolutional outline in any of the casts, though in some a degree of convolutional form can be found in the frontal and parietal regions. As I remember them, there is little in the occipital region, so that while one can determine the position of the rolandic fissure with some degree of accuracy in relation to the markings of the meningeal vessels in the dura, it is practically impossible to decide where the familiar markings on the cortical surface of the brain should be located.

I am at a loss to understand why the posterior area was marked off by the angle between the cerebellum and the under surface of the temporal lobe, because that is absolutely no index of the occipital area. The occipital area is limited cephalad by a line drawn between the incisura preoccipitalis and the parieto-occipital sulcus as it appears on the convexity; it therefore has no relation to the position of the cerebellum. The cerebellum in its relation to the cerebrum depends on the axis of the approach of the brain stem to the hemispheres.

When Dr. Casamajor and I were in London last summer, Mr. Pycroft, who has done so much work on prehistoric skulls, permitted us to examine the Rhodesian skull. This remarkable skull is complete except that the mandible is gone, and the squamosa is deficient on one side. Mr. Pycroft expressed satisfaction that that portion of the skull was gone as it permits investigation of the intracranial surface; no one would have had the temerity to cut an opening in a perfect skull. The skull is enormous, and the teeth splay out at an oblique angle, rather than being projected downward as they are in modern man; it is heavy because it is impregnated with metallic salts. An interesting point was the evidence of disease. The entire alveolar process was riddled with caries. The external lamina of the alveolar process covering the roots of the teeth was eaten away in many places. In the intact squamosa there was a round hole, the edges of which were fairly smooth and raised, as though there had been an epidural abscess in the region of the middle fossa which had gradually produced an osteomyelitis and escaped externally.

Dr. Weil: I took the measurements of endocranial casts only of prehistoric man, as Dr. Tilney was kind enough to allow me to copy from his collection. Dr. Casamajor's suggestion should be followed. I can only emphasize what Dr. Riley said, that one is not allowed to draw conclusions from the position of the meningeal arteries as to the position of gyri and sulci of the brain itself. The problem was not taken up from that point of view. The problem was to obtain certain relations between well defined parts of the casts, the mathematical sides of which could be compared. I expressed no opinion as to the position of the gyri or the sulci of the brains themselves. If I used the terms "occipital" or "frontal" I did not mean to express an opinion as to the size of corresponding parts of the surface of the brains of prehistoric men. I only wanted to indicate the relative position of the three lines constructed in

the casts. If we take the problem from this point of view and only compare such relations without going into discussions about the configuration of the brain itself, it may be possible to get some idea whether these endocranial casts are similar to one another or not.

A Case of Multiple Sclerosis with an Unusual Symptom. I. S. Wechsler, M.D.

This patient, a man, aged 24, presents two rather unusual symptoms. He had tuberculosis four or five years ago, and the lesion is healed. He had typhoid fever fifteen years ago. Otherwise the past history is unimportant. Two and a half years ago he became sick with his present condition. The first symptom was numbness of the left lower extremity. Following that he developed certain symptoms on which he dwells a great deal. He complains of a peculiar electrical sensation going down the spine and lower extremities. He gradually developed all the symptoms of multiple sclerosis: diplopia, spasticity, increased deep reflexes and the Babinski sign, clonus, absent abdominal reflexes, nystagmus, and occasional tremors. He shows one peculiar symptom on walking which he does not show while in bed. All movements are practically normal in bed. At one time it was thought that it might be a case of astasia-abasia, but the fact that the patient shows undoubted symptoms of multiple sclerosis dispelled that idea. On walking he has a tendency to whirl about from left to right. I had him walk on a lawn and he walked in a wide circle, always from left to right. Another unusual symptom is sudden myotonia on attempting to perform a quick movement. This occurs only in the left arm. If you ask him to bring slowly the tip of the finger to the tip of the nose, he can do it well, but if you ask him to do it quickly he stops as if he had a sudden myotonia, the arm becoming rigid. He cannot feed himself with that hand unless he does it very slowly. The third spectacular thing is that in the dark he can "light" an electric bulb with his hands.

All laboratory tests give negative results. The Bárány tests are negative. The fundi are probably normal, though there is a slight pallor of the left disk. There are no visual disturbances and no sensory changes. He speaks of having had some sensory changes, but there are none at present. He has no vestibular signs. Presumably, the symptom complex is cerebellar in nature, and yet he shows few, if any, cerebellar symptoms objectively. I do not understand the myotonic symptoms. I may add that he develops a similar stiffness of the limb from a sudden psychic or emotional upset.

Finally, I will demonstrate a more or less spectacular performance, which the patient attributes to the electric sensations on which he harps so much, namely, his ability to obtain a glow from an ordinary incandescent lamp.

DISCUSSION

Dr. H. A. Riley: I hesitate to express an opinion on a case which I have seen only in this cursory way; but it seems to me that the burden of proof rests entirely on the defense to prove that this is not a functional condition. I have never seen anything like this in cerebellar disease or in cases of multiple sclerosis. I have never seen it in animals with cerebellar ablation. The only thing I have seen like this is the effect of rapid rotation of guinea-pigs when, as a result of the turning, they rotate extremely rapidly on their long axis. I think the question of this being an organic symptom is open to considerable doubt.

The myoclonic cessation of movement also is open to some criticism; I would like to see this patient try to catch a ball which is dropped vertically so that he would have to catch it from the side, or some other test in which he might become interested, before I would be at all certain that the patient's condition is due to any myoclonic action on the part of the musculature.

I have nothing to suggest in regard to lighting the bulb, except that I would like to see Dr. Wechsler try it himself. As far as I could see, he did

not light the bulb; there was no glowing of the filament inside of the bulb. I think it was simple surface frictional electricity spreading over the surface of the frosted bulb and causing it to glow.

DR. LOUIS S. ARANSON: I would agree with what Dr. Riley said about frictional electricity, except that I saw the tungsten filament light up in the dark from where I sat. Besides the multiple sclerosis he seems to have also hysteria.

DR. S. PHILIP GOODHART: I have no inflexible opinion as to this case. To me it is not psychogenic in any of its phases. The rotary whirling with such force is unique in my experience. I am inclined to place the seat of pathology in the cerebellum.

DR. WECHSLER: I am glad that Dr. Goodhart said what he did; the reason I do not think this is of psychogenic origin is that this man has an organic disease of the nervous system; furthermore, I have been unable to discover a psychogenic origin as a cause for it.

In confirmation of what Dr. Riley said, the patient is an electrician; he is intelligent, but is given to emotional outbursts. Whether that is psychogenic or a true thalamic symptom I do not know; but it occurred to me that one might turn this around and assume a disorder of the nervous system for the ordinary astasia abasia; there may be an organic explanation for that. This might be preferable to thinking that the man has a pure hysteria. Is it not possible that with certain symptoms of chronic encephalitis (which we know are not hysterical) and with certain tics and turnings which are usually called psychogenic, one may assume from this case that the astasia abasia is really due to an organic lesion? The conception is not novel. Wilson has attempted to explain hysteria in terms of decerebrate rigidity. This is one possible explanation of the symptoms which are ordinarily designated hysterical.

DR. RILEY: I do not mean to imply that this case is entirely functional. I believe those two symptoms can be a functional superstructure on an organic basis.

TWO CASES CHARACTERIZED BY INVOLUNTARY MOVEMENTS. S. BROCK, M.D.

Case 1 is that of a boy, aged 19, who at the age of 9 developed involuntary movements. These began with blinking of the eyes, facial grimaces, stuttering, and twitches in the shoulders. For the last ten years he has shown a picture which has varied from time to time. There have been remissions and exacerbations. He shows a fairly *pure* type of involuntary movement, a myoclonic twitch or ticlike movement. These are fairly lightning-like muscular contractions involving the musculature of the upper half of the body, neck and head. The eyes close; the head is turned around rapidly; the jaw is moved up and down; the pectorals twitch. There is a laryngeal tic which interferes with speech. The affection does not involve the lower half of the torso nearly as much. I present this boy largely as a prelude to the next case, because of this pure type of involuntary movement. The neurologic status is entirely normal except for these movements. The intelligence quotient is low. Whether that is due to poor educational facilities or to poor mental endowment is not settled. I present him as a case of generalized tic, the *maladie de tic* of the French. I believe there is an organic basis to this disorder.

Case 2 is that of an unmarried girl, aged 24, who has had a rather checkered career since the age of 15. This may have some bearing on the diagnosis and prognosis. She was an orphan, ran away from school and joined a theatrical troupe. At the ages of 17, 19, and 21 she had abortions performed. In August, 1925, she acquired syphilis. Under observation at the Montefiore Hospital she has shown a considerable degree of emotional instability, so that she might well be regarded as a type of constitutional psychopathic personality. The other elements in the history are that, in 1918, she had what was called influenza. There is a history of diplopia recurring from time to time, but no

other postencephalitic phenomenon. She is hazy about an important point: some time in 1918, either before or after the influenza, she slept a great deal. She would fall asleep in the schoolroom and be scolded by the teacher. She recovered entirely from the influenza; but, in 1921, she had "tremors of the legs." They apparently disappeared, and the present condition began about October, 1926, following an operation for a diseased appendix and hernial repair. She began to shake; this came on acutely, and she has been presenting this group of involuntary movements ever since. I have seen her both at Bellevue Hospital and Montefiore Hospital over a long period of time. In contradistinction to case 1 she shows a veritable symphony of involuntary movement. If you start with the head you will note that it is thrown back in a myotonic ticlike manner; the jaws move from side to side; there are various clucking sounds made by ticlike movements of the tongue and mouth. At times, when the head is thrown back the eyes will rotate upward. The sternocleidomastoids, throwing the head from side to side and bringing it forward in a dystonic manner, seem hypertrophied under this constant activity. The upper extremities have a kind of pattern; there is a rather coarse rhythmic element to some of the movements reminiscent of Parkinson's disease. Often there are wild flinglike movements of the upper extremities in front of the chest. The movements of the neck are quite characteristic. She presents a fairly constant coarse rhythmic tremor; sometimes both hands are pronated. A choreoathetoid pattern is present in the hands. The speech is considerably interfered with. In walking, she walks on the outer sides of the soles, and the feet have an equinovarus posture. Curiously enough, when we examine the tonus, she shows the changing tonus (at times hypertonic, at others hypotonic) characteristic of a dystonic disorder. The attudinizing and dancelike gait are reminiscent of Huntington's chorea. Neurologic examination gives entirely negative results. Fragments of decerebrate rigidity are present; the feet in equinovarus, the pronated hands, the upper extremities in external rotation, the extended neck and head.

Dr. Kraus: It would be interesting to have her lie down on a flat surface; the picture changes a good deal.

Dr. Brock: In lying down, the dystonic foot comes out. The blood Wassermann reaction and spinal fluid findings are negative.

To give direction to the discussion, she is presented as a case of organic disease, of uncertain etiology, probably chronic encephalitis, perhaps syphilitic. I hesitate to name the disorder because it is too polydyskinetic.

DISCUSSION

Dr. Philip R. Lehrman: How soon after the operation did these movements appear?

Dr. Brock: They appeared during convalescence.

Dr. Kraus: I saw this patient on the wards of Bellevue Hospital. There was, at first, a little doubt whether or not the condition was primarly organic; after a short time that doubt was definitely and permanently dispelled. After seeing many cases of dystonia musculorum deformans, one has the impression that this girl belongs in that group. At Montefiore Hospital last year, five cases of chronic chorea were brought together at a conference; each case appeared a little different, once they were collected together; yet, each separately was definitely a case of chronic chorea. The same thing applies to this case. It looks different from other cases which we call dystonia musculorum deformans, but that does not mean that it should not be put under the caption of that disease.

Dr. Michael Osnato: In hysterical manifestations of any severity there are objective symptoms. I fail to recall at this moment any hysterical patient as disabled as this patient who did not show objective somatic sensory or

motor hysterical phenomena. I mean the anesthesias and the special sense disturbances, especially of the visual fields and the various auditory phenomena. They are absent in this case; that fact should receive more attention than it has. I find them present almost invariably, especially in post-traumatic cases. I agree with Dr. Kraus and Dr. Brock that this is an organic case, and that it can well be placed in the group of dystonia musculorum deformans. Until we know more about the pathology and the natural history of dystonia we ought to conserve the term and apply it only to that very definite group entity which Ziehen first described and Oppenheim later elaborated. There are cases such as this which do not exactly fit the classical picture, but which undoubtedly belong in that general group. All neurologists have observed patients who presented residuals of epidemic encephalitis when examined, yet who had never been ill enough to go to bed and in whom the history of the ordinary symptomatology of epidemic encephalitis was extremely difficult to elicit. In view of that fact I believe both cases may be looked on as probably postencephalitis.

Dr. George H. Hyslop: The case of the girl, together with others of its kind, leads to a dispute which is interminable. I have seen only one or two in whom one very important test had been applied; submission to the gamut of direct and indirect suggestive therapy. One might find psychogenic factors as the result of some sort of analysis. One cannot, however, prove these to be etiologic factors in a situation like this without a therapeutic result. I know of no organic pathology which would account for this girl's behavior any more than for what appeared in the boy with multiple sclerosis. These patients are in an environment which induces exhibitionism; they are all worse when under emotional stress, which is true in cases of either organic or psychogenic origin. There is much frontal lobe display in the girl's "hyperkinesia." So far as I know there is no tonus disorder. There is no real dyssynergy of the sort one can attribute to disease of the basal ganglia or of the cerebellar pathway.

Dr. Junius Stevenson: I was the first to see this patient. I do not question the probable organic basis. There is no question in my mind that there is a functional element. The movements are quite different from what they were at Bellevue. She then showed movements typical of the "Charleston" dance. While she was under observation and discussion various men made different suggestions; she developed movements suggestive of these things. I believe the movements are the result of suggestive examinations; I do not believe that the case belongs in the category of dystonias. I never considered her hysterical; almost all the movements are purposeful; the movements in hysteria are not. If this had been hysteria she would have been well before this. Suggestive therapy has been attempted; hypnosis was tried but it was found impossible to get her under. The girl has gone through three separate series of movements.

Dr. Russell MacRobert: I have seen the patient in case 2 before. In progressing across the room the movements have a certain rhythm, an almost musical rhythm. In walking up and down before us here the gait is actually the performance of a solo jazz dancer. Perhaps the psychoanalyst might discover that she was acting out a thwarted desire to be a famous dancer. Tonight she surely appears to be deriving pleasure from the exhibition. She proceeds back and forth in dangerous proximity to these desks, shakes off proffered assistance, misses everything and does not hurt herself at all. That is evidence of psychogenic motivation, is it not?

It would be impossible to demonstrate, as Dr. Osnato suggests, the presence or absence of corneal and pharyngeal anesthesia in a patient going through such constant contortions and spasms. There is probably some organic change in the nervous system; but I consider it incorrect to present this performance as a consequence of organic brain disease, and wish to register my opinion that the major manifestations are of a psychogenic origin akin to hysteria.

Dr. S. Philip Goodhart: The careful avoidance of objects in the patient's way, as she moves about, with apparent loss of control of coordination in motility, is observed often in patients whose deformities of motility are those involving the extrapyramidal mechanism. Indeed, I have often wondered at the agility of these patients with their violent motor defects in synergic control. There is much of the typical organic defect in the movements of the girl. The symmetrical involvement of agonist and antagonist groups of the shoulders is a not uncommon form of group involvement in postencephalitis. It is probably true that an emotional element in the personality make-up gives a psychogenic impress to the picture.

Dr. Wechsler: At this date one should go a little further than to diagnose hysteria by exclusion, and should not be content with saying that because a patient has not something therefore he has something else. Without discussing psychoanalysis, I think that it has afforded excellent insight into mental mechanisms; one should prove the psychogenesis of symptoms before saying a patient is hysterical. As long as that is not proved, it is not permissible to say that a patient has hysteria because the symptom looks like it. Seemingly purposive movements may be due to organic disease of the nervous system, and those movements can simulate hysteria. A pattern and seeming purposefulness do not necessarily imply hysteria. Every movement obviously follows a pattern; it depends merely on the functional level at which the disease occurs to bring out the complexity of the pattern.

The point raised by Dr. Kraus can, I think, be accepted only in part. There is no doubt that in dystonia there is dystonia, that is, constant change from hypertonus to hypotonus. He is correct in putting dystonia in the group of dyskinesias, but variations in tone are pathognomonic features. If anything is characteristic of dystonia it is posture. Dr. Brock and I described a myostatic form of dystonia wherein the patients who previously had had typical dyskinetic movements lost them and were left in a sort of "frozen" postural state against which the previous movements had been silhouetted, as it were. In every case one can see definite postures between movements, and it is those postures which seemed to us to characterize dystonia. In fact, we have seen patients go from the postural form of dystonia with few movements to a phase of violent dystonia, and vice versa. The term dyskinesia is a generic one, and while dystonia belongs in that group, just as do athetosis and chorea, the essential features of dystonia are disturbances in tone and posture.

Visceral Disorders Associated with Dystonia Musculorum in Epidemic Encephalitis. S. Philip Goodhart, M.D.

The purpose of this communication is to direct attention to certain features of the clinical syndrome dystonia musculorum deformans, especially, and perhaps only present, when the syndrome is a part of the pathologic process of epidemic encephalitis. The details of the observations, analysis and conclusions by Drs. Ralph Kaufman, Nathan Savitsky, and J. Robert Fried, resident internes and radiologists respectively in the Montefiore Hospital, will appear shortly. During the past decade, we have had under observation a large number of patients showing the syndrome of dystonia musculorum, both of encephalitic and of unknown origin. In the cases following the appearance of encephalitis of the epidemic type, not only were familiar disturbances of the somatic musculature observed, but visceral symptoms, suggesting involvement of the vegetative nervous system, accompanied the hyperkinesis and postural deformities in a strikingly large number of the patients. Just as formerly, much of the picture of motor distortion was erroneously conceived of as psychogenic; so, for a long time, were the disturbances of visceral control considered functional. It is to the credit of Drs. Kaufman, Savitsky and Fried that their timely observations and studies appear to confirm the view that the frequent and often severe participation of the vegetative nervous system in these cases

is part of the central organic process involving that area of the brain so commonly the seat of the pathologic process in encephalitis.

The most striking symptoms were those involving the gastro-intestinal tract, although disturbance of bladder control was not uncommon. The gastro-intestinal symptoms, intractable and uncontrollable vomiting, could not be explained on any organic basis after careful examinations in all directions. In two of the cases the paroxysmal attacks of vomiting led to extreme emaciation. One of the patients was given repeated hypodermoclysis and intravenous feeding to sustain life. In all cases these paroxysms would cease spontaneously.

In another instance, R. C., the patient originally shown as having symptoms probably of psychogenic origin and who later developed a characteristic dystonic syndrome, was, we have reason to believe, suffering from an organic disease. The pertinent feature is that the visceral symptoms presented shortly before the development of a typical dystonic syndrome were: sudden and extreme abdominal distention, obstinate constipation, and vomiting of such intensity as to lead to the diagnosis of an acute abdominal inflammation requiring surgical intervention. The patient was taken to the Jewish Hospital in Brooklyn; the visceral symptoms spontaneously subsided and the dystonia rapidly developed. There was a history of previous encephalitis. The patient has suffered since with intense seizures of protracted vomiting and of vesical retention.

Fluoroscopic studies revealed abnormal hypermotility of the stomach and esophagus in a number of the cases of dystonia with encephalitis as the etiologic factor; the observations were conducted with barium. The typical picture was that of a series of antiperistaltic waves which involved the entire esophagus; there was regurgitation of the barium and only later did some enter the stomach. The marked contractions of the stomach wall throughout the examination caused a reflux of the barium into the esophagus; the obstruction to the flow of barium appeared to be due to muscle spasm. In the case of another patient, shortly after taking the barium the stomach walls began to contract with such violence that the barium was shot upward toward the cardia. The observations were made by the resident and attending roentgenologists at Montefiore Hospital. Fluoroscopic examination of the bladder in several cases has proved unsatisfactory.

I have long been of the opinion that there is a close relation between the nervous centers of control and the physiologic function of striated and non-striated muscle and perhaps too of higher nerve control, both for nutrition as well as motility. Postmortem studies of a series of cases of muscular dystrophy by Dr. Globus and myself, published a few years ago, showed characteristic trophic changes in the cardiac nonstriated muscle in some of the cases. Perhaps the underlying pathology of the dystrophies lies in a trophic center not remote from those of kinesis in the ganglia of the forebrain.

DISCUSSION

DR. M. NEUSTAEDTER: Recently, Dr. Wimmer spoke here of a series of twenty-four cases of the syndrome of amyotrophic lateral sclerosis with the etiology of epidemic encephalitis. I know of no symptom or organic syndrome in neurology that is not encountered in association with epidemic encephalitis. Tonight the complex is that of dystonia musculorum. When discussing Dr. Wimmer's paper I stated that the encephalitis in amyotrophic lateral sclerosis, and I believe it is also true in dystonia musculorum, was merely an exciting cause that produced the symptom on an already existing point of minor resistance. Racially, these patients are mostly eastern European Hebrews. My two patients in the Central Neurological Hospital, two brothers and a sister I saw in Berlin, and other recorded cases seem to have some racial stamp, just as in amaurotic family idiocy. It would be worth while to reduce epidemic encephalitis to certain symptom-complexes, like poliomyelitis, that have a definite basis as the result of an inflammatory condition, and speak of these degenerative disorders, or whatever they are, that occur with encephalitis as

inciting causes. In the girl presented there is a possibility that either the encephalitis or the syphilis or both factors were the exciting causes that brought on these phenomena which appeared rather suddenly with remissions. That is a peculiarity. In the ordinary slowly progressive case of dystonia there are no remissions. Progress from symptom to symptom, from limb to limb is continuous.

Dr. Michael Osnato: It is clear from Dr. Goodhart's paper that he is talking about a syndrome of epidemic encephalitis and not about the extremely rare classical dystonia musculorum deformans, just as he might have discussed the parkinsonian syndrome in encephalitis. That must be kept in mind. He is pointing out that in this group of dystonic cases following epidemic encephalitis one may encounter a group of symptoms which are properly referred to the sympathetic nervous system; such symptoms are seen in other types of epidemic encephalitis cases without the dystonic picture. The value of Dr. Goodhart's paper lies in the demonstration that the dyskinesia occurs apparently not only in the somatic but also in the visceral musculature.

Dr. Charles Rosenheck: Functional superposition on an organic encephalitic basis is ruled out in the cases under discussion. In the vast majority of cases of encephalitis there is little gastric disturbance. Vegetative dysfunction is evidenced by abnormal weight increase, excessive sweating, polydypsia, and polyuria; but one rarely encounters vegetative disturbances such as Dr. Goodhart has described. I do not question the possibility of an organic central basis for them, but it seems to me that patients of this type, particularly in certain racial groups, are much disturbed by their tragic condition; perhaps this functional reaction is evidenced by the extreme gastric disturbances. I am not questioning the fact of the gastric upset, but is it not possible that it is merely a functional superposition? I want to stress the fact that gastric upsets of such marked severity are quite unusual in cases of epidemic encephalitis.

Dr. Moses Keschner: Dr. Goodhart made it clear that this is only a preliminary communication. I have had something to do with the work and can corroborate the statement that the observations have been peculiar. The whole neurologic staff is aware that vegetative symptoms are common in encephalitis, but these unique symptoms referable to stomach motility, to the bladder, and even to the uterus are so unusual that one is led to believe that there must be some relation between them and the disease itself. When we asked the radiologist to study these cases we asked him definitely whether the picture was or was not comparable with ordinary cardiospasm; he assured us that this is not the roentgen-ray picture observed in ordinary cardiospasm. This work requires much study. From the evidence obtained thus far we are not prepared to go on record as saying that vegetative nervous symptoms are part of the dystonic syndrome of encephalitis. It will be necessary to wait for postmortem evidence. I must emphasize again the peculiar form which these vegetative nervous system symptoms have assumed in these cases; they were so unique that, even taking into consideration the racial factor and all other factors enumerated by the other speakers, we feel the problem merits attention.

Dr. Walter Kraus: Last year, Dr. Perkins and I, at the meeting of the American Neurological Association, reported a case which we believe pointed to the thesis that the visceral nervous system exerts a trophic control over all parts of the body. As far as I know the only important structure of the body for which this has not been proved is the brain itself. Dr. Goodhart's presentation appears to me to furnish even more proof of this thesis. I believe that muscular dystrophy is a trophic disorder of somatic muscle. It is interesting that many of those who first described cases of muscular dystrophy believed this to be the fact. That concept died out, and later the idea that muscular dystrophy is a purely muscular disease came into vogue. The visceral nervous

system, as Dr. Goodhart showed some years ago, has trophic control over visceral muscle. He demonstrated a lesion of the heart muscle in muscular dystrophy. Dr. Goodhart tonight has shown that the visceral nervous system produces a very definite pathokinetic effect on viscera dependent on a pathologic state of the brain, which in this case happens to be epidemic encephalitis. In other words, there is both a trophic and a kinetic control of both types of muscle, somatic and visceral.

The puzzling feature to me is whether there is a trophic as well as a kinetic control of the gland cells. Of this I know nothing. In epidemic encephalitis the midbrain is profoundly affected. The kinetic control of the glands is profoundly affected; sweating, seborrhea and hyperacidity occur. Whether there is a normal as well as an abnormal trophic control parallel to the normal and abnormal kinetic control of glands is a subject worth considering.

DR. GOODHART: Dr. Neustaedter's remarks that the dystonias are by no means dependent etiologically on encephalitis alone are true. I do not contend that they do thus depend; in fact, as Dr. Tilney and others have maintained, it seems unjustifiable to assume that because one cannot find a recognizable etiologic or provocative factor in pathology, one must conclude that an inherent tissue defect, an abiotrophy, is the answer. I am not won over to the view that the dystonias, like other syndromes of central nerve pathology may result perhaps from influences of toxic or other destructive nature, and are not necessarily determined only by a faulty vital tissue capacity. That aspect of Dr. Neustaedter's criticism, however, is foreign to this presentation. The point made by Drs. Kaufman, Savitsky, and Fried is that apparently only in the dystonias of epidemic encephalitic etiology does one observe marked involvement of the vegetative nervous system. Dr. Osnato has properly emphasized the point of my communication, namely its purpose to bring out the association between dystonia of the type described and the vegetative nervous system.

Dr. Rosenheck's suggestion that these symptoms on the part of the vegetative nervous system may be psychogenic is the very bone of the contention that the present investigation endeavors to settle. The observations of the young men, on which this preliminary communication is based, to my mind definitely establish an organic basis for these symptoms.

Book Reviews

DISORDERS OF THE NERVOUS SYSTEM IN CHILDHOOD. By BRONSON CROTHERS, M.D. Price, $4. Pp. 242. New York: D. Appleton & Company.

This volume contains the personal experiences and thoughtful conclusions of an active practitioner. As the publisher announces, the book is monographic in form and clinical in presentation, like the others of this series on "Clinical Pediatrics." It is original, not in its facts, which are the well known data of neurology and psychiatry, but in its point of view. As the author puts it: "I am making no attempt to describe all the various syndromes due to lesions of the nervous system. Nor will I attempt to provide the intellectual tools for making elaborate neurological diagnoses. I feel that the important thing is to present a point of view. I believe that, in cases where damage to the central nervous system is suspected, only a few important questions arise.

1. Can it be arrested?

2. Is it inevitably progressive?

3. If the process is no longer active, does the physiological residue justify efforts at education?

The treatment of acute disease is a medical problem. The intelligent observation and classification of progressive lesions, for which treatment is not available, is a matter for experts in neurology or for students in the research laboratories. On the other hand, the intelligent management of children handicapped by fixed lesions of the central nervous system demands the most active attention from doctors, teachers, psychologists and parents.

In much the same way, in the so-called 'functional' diseases the doctor is one of a team whose job it is to educate the child. The problem is often difficult and its solution frequently demands concessions and disappointments. But the result if successful is worth the trouble.

If I can place before the medical profession this general conception of neurology and psychiatry as tools for the direction of education in a broad sense, I shall feel quite resigned to the proper disappointment which will be aroused by the cursory attention given to the syndromes which perpeuate so many of the names of great neurologists."

The first ten chapters discuss anatomy and physiology, neurologic examination, birth trauma, congenital anomalies, hereditary degenerations, infection, hydrocephalus, tumors, convulsions and cerebral palsies. In making this brief presentation the author has the great advantage of having approached neurology from the physiologic standpoint after a long training in pediatrics. He has written a book that is clinical in the best sense—it is practical, and carefully based on pathology and physiology. At times he is a little too impatient with the old-school neurologists. In fact, he has relegated the "Conventional Syndromes" to one short chapter at the end of the book. One admires and welcomes his clear physiologic expositions shorn of historical nomenclature, but one should remember that it was the painstaking descriptive work of the early clinicians and pathologists that made Crothers' functional synthesis possible.

The presentation is personal and direct. The author states the evidence (anatomic, physiologic, pathologic) and then boldly expresses his own opinion, telling specifically what should be done and what should be told to the parents. For instance after describing the mechanism of hydrocephalus he says:

"Several methods of approach are open. The simplest, and I believe a perfectly justifiable one, is to do nothing. On the whole the evidence is clear that even advancing cases sometimes become arrested. It is equally clear

that the study of a case involves danger. Even the removal of the fluid by puncture sometimes causes death or appears to cause increased production. The introduction of dyes or of air is by no means a procedure to be carelessly undertaken.

My own attitude is this: If the family feel that the condition, without interference, is intolerable and insist upon making use of every possible means of study, I am willing to go ahead. On the other hand, I feel that the chances of successful treatment are very slight and I am therefore unwilling to urge interference."

Chapters XI to XVII are concerned with psychiatry, behavior problems and educational possibilities. The point of view here expressed is most heartening, for the author rides no hobby and represents no cult. Although he modestly depreciates his psychiatric experience, it is evident that he has read widely and seen innumerable real problems in many homes. The chapter on "Methods of Approach in the Study of Mental Difficulties" is particularly commendable in its sanity. He urges the physician not to avoid mental problems because recent advances in psychiatry have made the technic appear difficult. In fact, it is emphasized that: "the family doctor in too many cases throws away therapeutic resources, which he has by virtue of his knowledge of his families, either because he is indifferent or because he has been over-awed by psychiatric and psychological vocabularies." Cases are described in which: "the whole problem resolved itself into a study of family relations and educational opportunities, and in which the situation demanded no technical psychology or psychiatry; yet it is hardly conceivable that the facts, upon which a solution could be based, could be collected by any one except a physician."

It is thus shown that the method of attack is not necessarily complex and technical; common sense and care can often do much. But the following points must always be kept in mind: "(1) it is by no means clear that the child is the one to be studied with most attention. He may be merely the casual consequence of a disturbed environment which needs investigation and correction. (2) No opinion worth giving can be given after perfunctory study. Of course, it is frequently possible to size up a situation in a very short time, but such snap diagnoses as 'silly mother,' 'absurb discipline,' etc., are not the basis on which treatment can be carried out without prolonged argument and detailed study. (3) Any abnormality or suspected deviation from normal which is preventing the child from getting on in a satisfactory way is of great importance. Furthermore, the mere fact that anyone thinks anything is wrong is important. A perfectly good child can be seriously injured if the family feel that he is unsatisfactory."

Such is the attitude and helpful simplicity of the book. The later chapters take up tics and habits such as enuresis and masturbation and the book ends with a plea for pediatricians to enter mental hygiene work. It is pointed out that pediatricians have done wonders in improving physical conditions and may well be proud of their efforts. Now they have the chance to enter work in mental hygiene almost at its inception. If they hold back they may easily find that they are left behind. The book ends with a selected bibliography and adequate index.

LES TROUBLES VASCULAIRES DANS LE TABES. By MAURICE BASCOURRET. Pp. 199. Paris: Jouve & Company, 1927.

It is a curious fact, says the author, that in spite of the syphilitic vascular conditions so often found in other diseases, tabetic patients are fairly exempt from cerebral hemorrhage, thrombosis and cardiac accidents. The author, therefore, studied seventy patients from the standpoint of the cardiovascular system. Ringing aortic second sounds were present in only two cases. Aortic insufficiency was present in four cases, and merely a systolic murmur at the aortic area in seven. A few cases of irregular rhythm were disclosed; tachy-

cardia, on the other hand, was unusually frequent, particularly in the later stages. This was not particularly dependent on the shooting pains. The oculocardiac reflex was normal. Palpitation was a frequent symptom, and, occasionally, painful sensations occurred in the chest, somewhat resembling and possibly caused by gastric crises. Objective signs of cardiac insufficiency, however, were rare; acute edema of the lungs and cardiac death were infrequent. The blood pressure was, as a rule, high. Over half the cases presented a systolic pressure of 170 or over, and very few presented hypotension. In the late stages, with asthenia and emaciation, blood pressure was apt to be low. Other things being equal, hypertension in tabes was correlated with age. No subjective disturbances seemed to be caused by this hypertension, and headaches, particularly, were absent. Vasomotor disorders similar to those found in other cases of hypertension were remarkably frequent. The blood pressure seemed rather unstable, and was not particularly affected by the pain.

Hemiplegia from hemorrhage or thrombosis was rather infrequent. A characteristic type of hemiplegia has been described by Fournier; it develops early and disappears. This is in contrast to the enduring, severe hemiplegia of sylvian thrombosis. The author was able to collect from the literature only forty cases of tabes complicated by persistent hemiplegia. Bulbar and spinal lesions likewise are infrequent; if they occur, they come early and tend to disappear. The peripheral vessels are also not subject to thrombosis. Postmortem examination of twenty-five brains showed only occasional pathologic alterations in the vessels, in spite of the hypertension and tachycardia, and in spite of the advanced age of many of the patients (in the seventh and eighth decades), there was little atheroma of the cerebral vessels. Hemorrhage into the brain was disclosed three times and softening twice in the twenty-five specimens examined; two more presented minute infarcts in the basal ganglia. These patients averaged 63 years of age.

Clinical study of certain patients revealed the fact that there was marked microsphygmia in tabes. This work was carried out with the Pachon instrument. It was more pronounced in the lower limbs, and did not show any relationship to arterial pressure. It was apt to be more marked in cases of long standing. On the other hand, "one may find in tabetic, especially at the beginning or in those fixed cases showing few symptoms or arthropathies, crises of local or general macrosphygmia co-existing with the development of arthropathies, spontaneous fractures or shooting lightning pains, or even spontaneously without apparent cause." The prevailing microsphygmia is revealed in the pallor, which is so characteristic of old tabes.

Vasomotor reflexes were studied in various ways. The hot bath gave hardly any color to the hands in cases of long standing, and the only frank reactions appeared in recent sufferers. The reaction was even more difficult to obtain in the lower limbs. The cold bath likewise revealed very little change, although it was a long time in many cases before certain patients recovered their normal cutaneous temperature.

In endeavoring to disclose further facts concerning the sympathetic system, the authors used various active principles, but they had to give up suprarenal extract because severe lancinating pains arose in certain cases. Pilocarpine likewise was apt to cause vomiting and diarrhea. The results obtained in the usable cases showed that sweating was difficult to provoke, especially in the lower extremities. The pilomotor reflex was exaggerated in certain cases. On the other hand, the reflex was often absent in advanced cases and was seldom found in the lower limbs. Scratching the skin seldom gave rise to a red line. The skin of a tabetic patient felt like that of a dead person. Occasionally, crises of bounding pulse were observed, sometimes in response to pain, sometimes without such connection. The most profound observation on such crises was reported by Pal. Flashes of heat, the outpouring of sweat, sensations of cold or of burning in the body and limbs are phenomena frequently recognized in tabes. Erythromelalgia, dermographism, livedo, acrocyanosis,

spontaneous ecchymosis and divers eruptions and edemas have been recognized. Secretory disorders are recognized in diarrhea, sialorrhea, and even galactorrhea. Exophthalmic goiter in tabes has been reported. Trophic lesions of the bones, joints and skin seem to follow more or less an increase in the pulsation of the vessels, and these trophic disorders are extremely frequent in tabes, provided a careful search is made. After the acuteness of the trophic lesion has passed, the pulse again becomes small.

The rest of the essay is a series of questions and attempts at answer, the answers sometimes being ingenious, sometimes rather far fetched; following this, some cases are reported in detail.

A Textbook of Psychiatry. By Arthur P. Noyes, M.D., of. St. Elizabeth Hospital, Washington, D. C. Pp. 333. New York: The MacMillan Company, 1927.

There is something attractive about this the latest textbook of psychiatry which is the product of several years of lecturing to nurses at St. Elizabeth Hospital. It shows a straightforwardness, simplicity and clearness which result from exposition of a subject to hearers who do not possess advanced training and who do not know the meaning of technical terms.

"It is recognized that mental nursing can no more be divorced from physical nursing than can the mind be separated from the body. Just as long as there is a body to be nursed so long will the problem be one not merely of physical care but also of human behavior." The mind is "that aspect of the organism which unifies its activities"; that aspect is found down through the animal scale to the ameba. Consciousness is awareness, the apex of the conventional triangle whose lower areas are lost personal memories, then archetypes or forms of thinking and feeling inherited by the race, and lastly instincts or inherited (racial) forms of action. The author passes from the neuron on page 4 to sublimation on page 22 at praiseworthy speed.

The mind at work employs over and over certain methods which are revealed plainly in mental disease. These methods have been adopted by all people as ways of dealing with a conflict within them between early, animal-like selfish tendencies and late civilized ethical standards. They are, in their chief forms, repression, compensation, symbolization and projection. Then there are the responses of extroversion and introversion, fixation fantasy and dissociation. In studying psychiatry the nurse will get more from an understanding of these methods, by which her own mind works, than from "the knowledge she secures of a special branch of nursing."

Chapters IV to XXVI present adequately and conventionally the major psychoses. In the chapter on manic-depressive psychoses excellent hints on nursing stand out, but there are statements concerning the nature of the disease which cannot be fully accepted. "Manic-depressive psychoses represent the reactions of the entire personality to difficult and painful problems and situations which have an ultimate origin in the unconscious." An example of manic behavior which should become classic is found here. The patient went to his mother's funeral and returned to the hospital wearing a broad band of crepe on his sleeve, a red tie, purple stockings and a gay colored waistcoat. The excellent chapter on the psychoneuroses treats them as "compromises which neither offend society nor damage the personality" as do the psychoses. In chapters on psychiatric nursing in general there is proper recognition of the high type of person needed, intelligent, not sentimental, resourceful and observant, with a discussion (not too flattering) of the possible reasons for the nurse's choice of her profession.

The whole book tends to make the nurse understand that she and the patient are made of the same clay, inherit the same patterns of thinking, feeling and acting, and can work out a more civilized answer to the problems which confront both of them though in different degrees.

MIND AND HEREDITY. By VERNON L. KELLOGG. Price, $1.50. Pp. 108. Princeton, N. J.: Princeton University Press, 1923.

This book contains a series of short connnected essays concerning the development of the mind, each chapter being more or less complete in itself but bearing a definite relationship to the others. The instinct mind of insects leads to the subject of tropism, reflexes and mechanistic qualities, in an endeavor to give factors of inheritance the large place they deserve in the development of the mind. The author leans definitely to a materialistic philosophy, for he says: "We have derived our body and its inherent capacities, physical and mental, by slow evolution from other early lower kinds of animals. We . . . find our mind a function chiefly of our physical nervous system." And yet, like all philosophers, he finds many phases of mind which cannot be definitely explained on a materialistic basis.

The subject of the inheritance of acquired characteristics begins and ends as a question, but the influence of training and environment is given the large place that it merits. The author agrees with Galton that minds are all determined as to character and capacity at birth, and that environment merely acts on a favored soil. He recognizes clearly the complementary function of heredity and environment, and says: "Both are necessary to our being at all."

Intelligence testing is reviewed and given a limited, but nevertheless valued, place in the estimation of the mind. In considering the national phases of immigration, the author frankly deplores the low intelligence of the majority of immigrants. He says: "The recent samples of all the races have been poorer than the earlier ones, and the samples of Southern and Eastern European peoples have been poorer than the samples of the Northern and Western peoples." He states, further, that the average intelligence of a considerable fraction of the immigration population is distinctly below that of the native population, and that the intelligence of millions of immigrants is markedly below that of the native born population.

Educational methods are analyzed, and stress is laid on the need for more individual training. The mass methods of teaching—characteristic of all schools in this country, including the universities—fail to develop, as they should be developed, the varying types of mind. An opportunity should be given every student to make the most of his inherent possibilities. It seems to be the objective in all modern schools to cater to the minds of limited possibilities and neglect those of greater capacity.

Near the end of the book, the author says: "With direct inheritance of mental capacity, with the inheritance of emotions and temperament, and with the inheritance of differences in the functioning of the ductless glands whose secretions powerfully affect both emotions and intelligence, we have an imposing array of inherited factors in mental and nervous make-up, in a word —mind."

The book is clearly and entertainingly written.